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TABLE OF CONTENTS

Page
Foreword 8
D. melanogaster
  Stock lists
    United States
      Ames, Iowa 37
      Austin, Texas 37
      Baltimore, Maryland 37
      Berkeley, California 38
      Bloomington, Indiana 24
      Chapel Hill, North Carolina 39
      Chicago, Illinois 40
      Cold Spring Harbor, New York 9
      Columbia, Missouri 40
      Columbus, Ohio 40
      Evanston, Illinois 42
      Kingston, Rhode Island 42
      Knoxville, Tennessee 42
      Los Angeles, California 42
      New Haven, Connecticut 43
      New Orleans, Louisiana 45
      Pasadena, California 15
      Philadelphia, Pennsylvania 45
      Salt Lake City, Utah 47
    Foreign
      Argentina 50
      Australia 50
      Austria 51
      Belgium 51
      Canada 51
      France 51
      Germany
        Berlin-Buch 52
        Berlin-Dahlem 52
        Göttingen 53
        Hamburg-Eppendorf 53
        Heidelberg 54
        Neustadt, Schwarzwald 54
      Great Britain
        Bayfordbury, Hertford, England 54
        Birmingham, England 55
        Edinburgh, Scotland 55
        Glasgow, Scotland 57
        Harpenden, Herts, England 57
        London, England 58
      Italy
        Milano 58
        Pavia 59
      Japan
        Kyoto 60
        Misima, Sizuoka-Ken 61
        Osaka 62
        Sapporo 62
        Tokyo 62, 63
      Netherlands 63
      Norway 64
      South Africa 65
      Spain 66
      Sweden 66

Switzerland ... 68

New Mutants

Report of Arvelighetsinstituttet, University, Oslo ... 69
Report of Instituto de Genética, University of Buenos Aires ... 69
Report of Istituto di Genetica, University of Milan ... 70
Report of M. M. Green ... 70
Report of P. T. Ives ... 70
Report of George Lefevre, Jr. ... 71
Report of Helen U. Meyer and Margaret Edmondson ... 71
Report of George H. Mickey ... 74
Report of Sidney Mittler ... 74
Report of E. Ohnishi ... 74
Report of S. Rosin ... 75
Report of H. Slatis ... 75
Report of F. H. Sobels, Elze C. Boterenbrood, J. Faber, and F. J. Oppenoorth ... 75
Report of Bentley Glass ... 76
Report of A. J. Bateman ... 77

Linkage Data

Report of R. Milani ... 79
Report of E. Ohnishi ... 79

Other Drosophila Species

Stock lists ... 80
New Mutants ... 94

Notes and News

Research Notes

Anders, Georges Pleiotropic effect of lozenge clawless ... 98
Anders, Georges Pleiotropic pattern of the lozenge-mutant in *D. pseudoobscura* ... 98
Asahina, Kazuo Studies on the Taxis of *D. melanogaster* ... 98
Basden, E. B. Drosophila in Scotland ... 99
Bird, Myrtle J. Chemical mutagenesis ... 100
Brunetto, Anna, and Frumento, Luigia Salivary chromosomes of *D. ambigua* ... 100
Burdette, Walter J. Incidence of tumors in different strains of Drosophila ... 101
Burla, Hans Drosophilids of the Ivory Coast (French West Africa) .. 102
Buzzati-Traverso, A. A. Interspecific crossings in the *affinis* subgroup ... 102
Buzzati-Traverso, A. A. Interspecific crossings in the *obscura* subgroup ... 102
Buzzati-Traverso, A. A. Interspecific crossings in the *Pholadoris* subgenus ... 103
Buzzati-Traverso, A. A. Natural selection under increased mutation pressure in *D. melanogaster* populations ... 103
Carson, H. L. Interfertile sympatric sibling species within *D. bocainensis* Pavan and da Cunha 1947 ... 103
Cooper, K. W. The Chapter on Spermatogenesis in *Biology of Drosophila* ... 104
daCunha, A. Brito, Brncic, D. J., and Salzano, F. M. Comparative study of chromosomal polymorphism in populations of tropical Drosophila ... 104
Dale, Ernest E. Differential mortality ... 104
Dresden, D., and Oppenoorth, F. J. Selecting strains resistant to gamma-HCCH (hexachlorocyclohexane) ... 105
Edmondson, M. Interchange of eye and antennal tissue during development ... 105
Epling, Carl, and Mitchell, Donald F. A previously unrecorded gene arrangement of *D. pseudoobscura* in southern California ... 105

Faber, J. and Sobels, F. H. A new imaginal ring in the mid part of the hindgut 105

Fujii, S., and Kawabe, M. On the development of some "bristle genes" in the pupal stage of *D. virilis* 106

Gowen, John W., and Stadler, Janice Irradiation effects on viability of *D. melanogaster* 106

Imaizumi, T., and Kimoto, Y. Cytoplasmic constituents of eggs in early developing stages, and their staining properties 106

Ives, P. T., and Evans, Alice T. A probable simultaneous double mutation in the Cy sp^2 chromosome 107

Janzer, Wolfgang Studies on cave animal characteristics 107

Judd, Burke H., and Lefevre, G. X-ray-induced dominant mutation in *D. melanogaster* 108

Kikkawa, H. Effects of 3.4-dihydroxykynurenine on pigment formation 108

Koske, Thea A new species hybrid in the *obscura* group 108

Lewis, E. B. Additions and corrections to the cytology of rearrangements 108

Lindsley, D. L. An X chromosome specifically deficient for the nucleolus organizing region 109

Lüning, K. G. X-ray-induced mutations in different stages of spermatogenesis 109

Mainx, F. Structural variety in wild populations of European species of the *obscura* group 110

Makino, Sajiro, and Kanehisa, Takeharu A preliminary survey of the geographical distribution of Drosophila in Hokkaido 110

Makino, Sajiro, and Kanehisa, Takeharu A monthly survey of Drosophila in the City of Sapporo, Hokkaido 111

Makino, Sajiro, and Takada, Haruo A monthly survey of Drosophila in the vicinity of Otaru City, Hokkaido 111

Makino, Sajiro, and Kanehisa, Takeharu Some notes on a heritable tumor found in *D. virilis* 112

Meyer, Helen U. Evidence of the unsplit condition of interphase chromosomes 112

Mickey, George H. Origin of a new R allele in mosaic *D. melanogaster* 113

Mickey, George H., and Blount, Jerry Somatic polyploidy in *D. melanogaster* induced by cold shock 114

Mickey, George H., and Di Paolo, J. A. Lethals induced in Drosophila by combined action of urethane and H_2O_2 114

Mickey, George H., and Sturtevant, F. M., Jr. Failure of phenol to produce lethals in Drosophila 114

Miller, D. D. Mating behavior in *D. athabasca* and *D. narragansett* 115

Mittler, S. Variation of the penetrance of tu^{50j} when reared on yeasts that do not require vitamins or amino acids 115

Moriwaki, D., Okada, T., Ohba, S., and Kurokawa, H. Drosophila species belonging to the "*obscura*" group found in Japan 116

Mossige, Jeanne Two new jaunty mutations 116

Muller, H. J. Detection of mutations in the second chromosome by use of the "sifter" stock 117

Muller, H. J. Homosexual copulation in the male of Drosophila, and the problem of the fate of sperm of males isolated from females . 118

Muller, H. J. Localization of Y:bw$^+$ insertion and cr-u sterile (crs) 119

Muller, H. J., and associates Ultraviolet induction of mutants at loci at which spontaneous mutants are known 119

Nakamura, K., Immaizumi, T., and Kitazume, Y. Amino acids in *D. melanogaster* 120

Nolte, D. J. Secondary genic products 120
Novitski, E. Autonomy of sterility of transformed females 121
Novitski, E. The compound X chromosomes 121
Novitski, E. Useful derivatives of the X·Y chromosome 122
Oftedal, Per Genetics and histogenesis of a new tumor, tu(2)49k . 122
Ohnishi, E. Bilateral asymmetry and correlative expression of wing and hind leg in Bd^{491} 123
Ohnishi, E. Tyrosinase activity during puparium formation in *D. melanogaster* 123
Oster, Irwin I. Accentuation of distinctions in larval Malpighian coloration by the feeding of riboflavin 124
Oster, Irwin I. An analysis of ultraviolet-induced lethals due to gene mutation 124
Prevosti, A. The vti and vli characters in a wild population of *D. subobscura* Collins 126
Ratty, Frank J., Jr. Lethal coverage with short duplications ... 127
Sattel, Walter Running and hopping abilities of *D. melanogaster* . 127
Scossiroli, R. Relation between successful interspecific crossings and gene arrangements 127
Semenza, L., and Barigozzi, C. Chromosomes of *Aphiochaeta xantina* 128
Singleton, J. R., and Zimmering, S. Interchromosomal interference in *D. melanogaster* 128
Sobels, F. H., Kruijt, J. P., and Spronk, N. Lethality due to combined action of the genes Dichaete and eyeless-dominant 128
Spiess, E. B. Recent collections in New England 128
Spiess, E. B., Ketchel, M., and Terrile, B. A. Physiological properties of gene-arrangement carriers in *D. persimilis* 129
Spurway, H. 130
Tattersfield, F., Kerr, R. W., Taylor, J., and Kerridge, J. R. Resistance to the toxic effects of DDT 130
Ulrich, Hans Killing of Drosophila eggs by partial X-raying 130
Ulrich, Hans Sensitive periods and egg-regions in production of the modification "abnormal abdomen" by X-raying eggs of *D. melanogaster* 131
Valencia, J. I., and Valencia, R. M. The ineffectiveness of extra heterochromatin in influencing mutation rate in the female 131
von Brandt, H., and Höhne, G. Mutagenic action of some chemicals. 132
Waddington, C. H. Selection of the genetic basis of an acquired character 133
Wallace, Bruce, and Demerec, Rada A test for translocation mosaics in Drosophila sperm exposed to nitrogen mustard aerosol. 133
Wette, Reimut Production of phenocopies by chemical substances .. 134
Yoshida, Y. "Conditioned lethal" 134

Technical Notes

Anders, Georges, and Schnitter, Marco A method of mass investigations in Drosophila eggs 135
Clancy, C. W. "Seeding" cultures with Fleischmann's New Dry Yeast 135
Green, M. M. Rapid preparation of cornmeal-agar medium 135
Herskowitz, Irwin H. Grid-smear technique for electron microscopy of salivary-gland chromosomes 135
Herskowitz, Irwin H., and Burdette, Walter J. Preparation of permanent aceto-orcein smears 136
Mittler, Sidney Medium for rearing yeasts that do not require amino acids or vitamins 136
Rosin, S. The position of the wings of killed drosophilae 136
Stone, P. C., and Zimmering, S. An effective mite control 137
Wallace, Bruce Estimating the size of experimental Drosophila populations 137

Wilson, L. Deterioration of brewers' yeast as a factor in comparative growth studies on Drosophila 138
Personal and Laboratory News 139
Materials Requested or Available 140
Drosophila Bibliography 141

Directory

Geographical 155
Alphabetical 176

FOREWORD

DIS continues to grow larger with each issue, despite the fact that many U.S. stock lists are now being omitted. This can be interpreted not only as an indication of the continued interest of Drosophila workers in this cooperative undertaking, but also as evidence of an actual growth and expansion of Drosophila research in recent years. Nineteen new laboratory listings have been added in the Geographical Directory of DIS-25. Less than half of these are accounted for by old contributors' moving to new locations; and the others represent new inceptions of Drosophila work. Each year, too, a greater volume of material is received from the individual contributors.

DIS is run on a cooperative basis, and participation is required of any laboratory or a research worker who wishes to receive copies. The suggestion has been received from several sources that the issues be made generally available on a subscription basis. In this connection it has been pointed out that certain sections of DIS, particularly the research notes and to some extent the directory and bibliography, are of interest to other geneticists not working with Drosophila, and should be made available to them. I feel, however, that if DIS were available by subscription it would become "another journal," and would lose the unique quality of a cooperative undertaking and all the advantages that go with it. The editor could no longer require that each laboratory contribute material to current issues; and it seems likely that the contents would thus suffer in up-to-dateness and accuracy. Although it is true that Drosophila workers are now sending in a larger number of brief reports about their current work, this seems to suggest the need of a journal where such material might be published, rather than the desirability of circulating DIS more widely. I do not feel that the solution of this problem is to be found in DIS.

Irwin H. Herskowitz has completed his bibliography of Drosophila literature from 1939 through 1950, and the manuscript has been accepted for publication by the Imperial Bureau of Animal Breeding and Genetics, which in 1939 published H. J. Muller's Bibliography of the Genetics of Drosophila. Herskowitz is interested in keeping his literature files up to date, and has agreed to edit the Bibliography section of DIS, beginning with this issue, combining the reports sent in to DIS by Drosophila workers with the other references that he collects.

The material of this issue was arranged by Agnes Fisher, typed by Mrs. Ethel P. Burtch, proofread by Mrs. Hilda H. Wheeler, and mimeographed by Mr. Leslie E. Peckham.

M. DEMEREC

Cold Spring Harbor
December, 1951

STOCK LISTS

Key to abbreviations:

(1) number in parentheses after Df, In, etc., indicates the chromosome or chromosomes involved in rearrangement
___ indicates attached X's or attached X-Y
X^c closed X
Df deficiency
Dp duplication
In inversion
T translocation
Tp transposition
/ separates two homologous chromosomes
; separates two nonhomologous chromosomes, and X from Y
, separates aberration from mutants carried with it
& is used to indicate two components of the $ requiring selection
The letter $\underline{l}$ is underscored whenever necessary to distinguish it from the figure 1, as in lethal and when $\underline{l}$ indicates December.

COLD SPRING HARBOR: CARNEGIE INSTITUTION OF WASHINGTON

Wild Stocks

1 Amherst-34
2 California-c (In(3R) P, ca/$\underline{l}$(3)c)
3 Canton-Special
4 Crimea
5 Florida-19 (inbred)
6 Formosa, Japan
7 Kyoto, Japan
8 Lausanne-Special
9 Oregon-S
10 Salta, Argentina
11 Seto, Japan
12 Swedish-b-6 (Swedish-b cleaned of inversions)
13 Tuscaloosa, Ala.
14 Urbana-Special
15 Woodbury, N.J.

Chromosome 1

17 amx/C$\underline{l}$B
18 Ax
19 B
20 BB
21 bb Y^{bb}
22 Bg B/In(1)AM
23 bi ct^6 g^2
24 bo
25 br
26 Bx^3
27 car
28 car bb
29 cm
30 co
31 ct v dy g f/In(1) A^{99b} sn^{3lf}
32 ct^n g^2
33 dd^2
34 dm/Cl, y Hw
35 dow/dl-49, y Hw m^2 g^4
36 ec ct^6 v g^3/C$\underline{l}$B
37 ec ct^6 s car/C$\underline{l}$B
38 ec dx
39 Ext/dl-49, y Hw m^2 g^4
40 f
41 f B/$\underline{y}$
42 f B^{i}B^i/$\underline{y}$
43 fa
44 flp
45 fo
46 fu^g/$\underline{\text{y v f car}}$
47 g^2 pl/C$\underline{l}$B
48 g^2 ty/$\underline{y}$
49 g^2 ty/Bbb
50 gg^2/dl-49, y Hw w lz^s
51 gt w^a/$\underline{\text{gt bb}^{11}}$
52 gt bb^{11}/C$\underline{l}$B
53 kz/C$\underline{l}$B
53a kz
54 $\underline{l}$(1)7 dl-49, y Hw^3 m^2 g^4
55 $\underline{\underline{l}^{J1}\ \text{sc}^{J1}/\text{l}^{J1}\text{sc}^{J1}}$ dl-24 (Muller)
56 lh B car bb/$\underline{y}$
57 m
58 M(1)o f/In(1)AM
59 na/sc^8 dl-49 y B w^a lz^s
60 ny/C$\underline{l}$B
61 oc ptg^3/C$\underline{l}$B
62 od car
63 ov
64 peb v
65 pn^2
66 ptg^3 pn^2
68 ras^2
69 rb cx
70 rg
71 rst^2/dl-49, y Hw m^2 g^4
72 rux/dl-49, y Hw m^2 g^4
73 rux^2
74 sbr/$\underline{y}$
75 sc cv v dwx/dl-49 y Hw m^2 g^4
76 sc cv v f
77 sc ec cv ct^6 v g f/ClB
78 sc^2 pn/$\underline{+}$
78a sc t^2 v f tu car ♂ & y f ♀ ("scart")
79 sc^{10} w^a
80 scp t

81 sd mc
82 svr w^{a}
83 sw
84 sy
85 sx vb^{2} sy/In(1)AM
86 t^{2} v f
87 tbd
88 tw
88a tw/Cl, y Hw
89 un^{4}
90 v
92 vs
93 w
94 w fa

95 w n f
95a w m f/ClB
96 w ct^{6}
97 w ec
98 w spl
99 w^{a}
100 w^{bf} f^{5}
101 w^{bf} lz/ClB
102 w^{bl}
103 w^{ch} wy
104 w^{co}
105 w^{e}
106 w^{e} sn/ClB
107 w^{e2}

108 w^{i} vb
109 w^{sat}
110 w^{t} fw
111 w; We/CPL CPR
112 y
113 y ac v
114 y ec v wy^{2}
115 y f
116 y fa^{n}
117 y pn
118 y sc
119 y sc w
120 y w
121 y^{2} dvr^{2} v f car/ClB
122 y^{2} wy^{2} g^{2}

Chromosome 2

123 ab^{2}/T(Y;2)E
124 abr/Cy, hk^{2}
125 al b c sp^{2}
126 al b pr cn vg a sp^{2}/
Cy, L^{4} sp^{2} (albasp)
127 al sp b/Ins(2L+2R)
Cy, S^{2} E-S
128 al dp b pr c px sp/
Cy pr (all)
129 al S ho/Cy
130 ap^{4}/Cy
130a ap^{4}/Rvd
131 b arch l(2)C/Cy,
L^{4} sp^{2}
132 b el
133 b gp a/Cy-RNS
134 b j
135 b rd^{s} pr cn
136 bat/Ins(2L+2R)Cy, S^{2}
137 Bl/esc
138 blo
139 blt
140 bw
141 c wt px
142 cg c/U
143 ch
144 cl
145 cn
146 d b/Cy, pr
147 dp
148 dp^{T}In(2L+2R)Cy S^{2}
E-S
149 dp b cn c a mr/Cy
150 dp b pr c px sp
150a b (pr) c px sp^{2}
151 dp pr px/Cy, pr
152 ds ft dp^{V2} l(2)M b pr/
Cy, dp^{2}
153 ds S G b pr/Cy, L

154 ed Su^{2}-dx
155 el
156 ex
157 fes lt/Cy, al^{2} lt^{3}
L^{4} sp^{2}
158 fj px sp
159 fr sp/Cy, pr
160 fr^{2} wt/Cy
161 Grv/Cy, dp^{2}
162 hk
163 ho
164 hy a px sp/T(2;3)
S^{M}, Cy
165 J/In(2L)+, l(2)B
166 J^{34e}
167 Jag
168 kn
169 L^{2}
170 L^{5}
171 lgl cn bw/Cy cn bw
172 l(2)H L^{2}/Cy, dp^{2};
(w)
173 ll^{2}
174 lm/Cy, S^{2} dp^{2} E-S
175 lt std/Cy, sp^{2}
176 ltd
177 M(2)173/Cy al^{2} lt^{3}
L^{4} sp^{2}
178 M(2)l^{2}/Cy, L^{4} sp^{2}
179 M(2)z/In(2L)t,
l(2)R
180 mi sp/Pm^{2}
181 mr bs^{2}/Cy, sp^{2}
182 net ed Su^{2}-dx
183 nw^{2}/Cy
184 Pfd+Ins(2L+2R)Cy, S^{2}
185 pi/Cy pr
186 Pin

187 pr en
188 pu
189 pys
190 rdo^{2}
191 rh
192 rl
193 rn l(2)rn/Cy
194 S Sp ab^{2} ltd/NS,
px sp
195 sca
196 shr bw^{2b} abb sp/
Cy, sp^{2}
197 Sk b/In(2L)NS
198 sm px pd/Cy, al^{2}
L^{4} sp^{2}
199 stw^{3}
200 Su-H whd l(2) Su-H/
Cy, al^{2} lt^{3} L^{4} sp^{2}
201 tk sf^{2} abb
202 tkd/Cy, al^{2} L^{4} sp^{2}
203 tkv
204 trm-Cy
205 vg

Chromosome 3

206 aa h
207 app
208 bul
209 bx^{34e}/Payne, Dfd ca
210 c3G
211 cd
212 cmp ca/In(3R)c, e
213 cp
214 cp p^{p}
215 cu kar
216 cur
217 cv-c
218 In(3R)Cyd
219 D^{3} H/Payne
220 Dfd/Cx, D
221 Dl^{3}/In(3R)C, e
222 dv/Mé
223 e^{4} wo ro
224 ell
225 e^{s} cd ro cmp ca/
Xa, ca
226 eg^{2}/Dfd
227 gl^{3}
228 Gl/Payne
229 Gl Sb/LVM
230 in p^{p}
231 jv Hn^{r} h
232 jv se
233 Ly/D^{3}
234 Ly Sb/LVM
235 M(3)/In(3R)C, e
l(3)e
237 M(3)w/In(3R)C, e
l(3)e
238 ma fl
239 mah
240 obt
241 pb/Cx, D
242 p^{p} by Sb^{Spi}/In(3R)C,
l(3)a
243 Pr/In(3R)C, e
244 Pt/Xa, ca
245 pyd
246 R/Mé
247 ra
248 ri p^{p}
249 rp/Payne, Dfd ca
250 rs^{2}
251 ru h th st cu sr e^{s}
ca(ru-cu-ca)
252 ru h th st cu sr e^{s}
Pr ca/T(2;3)Mé
253 ru h th st p^{p} cu sr
e^{s} (res)
254 ru^{g} jv se by/Mé
255 ry
256 se e^{ll}
257 se
258 se rt^{2} th/Mé
259 se ss k e^{s} ro
260 se ss ro
261 Ser/In(3R)c, e
l(3)a
262 snb
263 sr sed
264 ss bx
265 ss bxd k e^{s}/X^{a}
266 ss^{a}
267 st
268 st E-Mg e^{s} M g/
T(2;3)Mé
269 st sr e^{s} ro ca;
tu 36a
270 su-ve ru ve h th
271 th cu sr e^{s} ro ca
(theca)
273 th st pb p^{p}/Cx, D
274 th st W p^{p}
275 tt wo
276 tx
277 ve
278 W

Chromosome 4

279 ar/ey^{D}
280 bt
281 bt^{D}/ci^{D}
282 ci ey^{R}
283 ci^{D}/ey^{D}
284 ey^{2}
285 gvl ey^{R}
286 Mal; pr
287 Scn/ey^{D}
288 spa
289 sv^{n}
290 Cat/ci^{D}

Multichromosomal

291 Cy/Pm, ds^{33k}, H/C, Sb
291a Cy al^{2} lt^{3} L^{4} sp^{2}/Pm;
Cx, D/In(3R)Mo, Sb
292 sn^{3}; Mw/Payne
293 su-b; b pr c
294 v; st
295 y; bw; e; ci ey^{R}
297 y; bw; e; ci ey^{R}

Closed-X

298 X^{c}, y/f B

Deficiencies

299 Df(1)N^{8}/dl-49 y Hw m^{2} g^{4}
300 Df(2)bw^{5} sp/Cy, dp
303 Df(2)Px^{2}/Cy, L^{4} sp^{2}
304 Df(2)vg^{D}/U
305 Df(2)vg^{S}/Cy, al^{2} lt^{3} L^{4} sp^{2}
306 Df(3)Vn/In(3L)P, gm

Duplications

307 Dp(1;f)$135^{y^{2}}$; In(1)sc^{8}, Df(o+ac) w^{a} sc^{8}

Inversions

308 In(1)AB/y v f
309 In(1)A^{99b}
310 In(1)dl-49, v^{0} f
311 In(1)dl-49, y fa^{n}
312 In(1)rst^{3}, rst^{3} car bb
313 In(1) sc^{4}, y sc^{4}
314 In(1)sc^{8}, w^{a}
315 In(1)w^{m4}
315a sc^{S1} B InS w^{a} sc^{8} (Muller-5)
316 In(2LR)Gla/Cy pr
317 In(3L)pers

Translocations

318 XY', g^{2} By"/y/Y" (Stern)
319 T(1;2)7/ClB
320 T(1;2)A124/y v f car
321 T(1;2)Bld/ClB
321a T(1;3)w^{Vco}/y w bb?/Cx; D
322 T(1;4)w^{m5} (het ♂; hom ♀)
323 T(Y;2)J ltd/ab^{2}
324 T(2;3)P/Cy
325 T(2;3)S^{M} Cy/vg^{nw}
326 T(2;4)d/Cy, pr

Unanalyzed

328 Virus? CO_2-sensitive e (l'Heritier)

Special Stocks

329 Df(1)260-1(0+sc) Df260-1 s/dl-49, y Hw m^{2} g^{4}
330 Df(1)260-2(0+ac) Df260-2/Dp 118
332 Df(1)260-10 (Df bands 1-2), y (hom)
333 sc260-14 (In) (hom)
334 sc260-15 (Tl;3)
335 sc260-17 (Tl;2L) (hom)
336 Df(1)260-19 (Df bands 1-2), g (hom)
337 sc260-20a (Tl;4) (hom)
338 260-20f Dp(y^{+}) y sc
339 sc260-22 (In) (hom)
341 260-25b Dp y sc
342 260-25c Df(y^{-})y Hw w
343 260-27 Dp(1)y sc/y sc Dp
346 sc260-29 (complex Tl;2L;3L) sc/y sc w
347 260-31 y ac 260-31 (Tl;2L) y ac/y dl-49
348 w258-11 (Df w) y w258-11/dl-49, y Hw m^{2}
349 w258-14 (Df w) y w258-14/dl-49, y Hw m^{2} g^{4}
350 w258-18 (mottled w, rst; Tl;4) y w258-18/w & w
351 w258-21 (mottled w, fa dm; Tl;4) y w258-21/dl-49, y Hw m^{2} g^{4}
354 w258-36 (mottled w; Tl;2R) y w258-36/w & w
358 w258-42 (Df w) y w258-42/dl-49, y Hw m^{2} g^{4}
359 w258-43 (Tl;4) y w258-43/dl-49, y Hw m^{2} g^{4}
361 w258-45 (Df w) y w258-45/dl-49, y Hw m^{2} g^{4}
362 w258-46 (Df) y w258-46/w
363 w258-48 (Df w) w258-48/dl-49, y Hw m^{2} g^{4}
364 w258-52 (In) w rst/dl-49, y Hw m^{2} g^{4}
368 N25/dl-49, lz^{s}
370 y N38/dl-49
371 N264-2a y N264-2/dl-49, y Hw m^{2} g^{4}
373 N264-7a (In) N264-7 sn^{3}/dl-49, y Hw m^{2} g^{4}
374 N264-7b (In) N264-7 sn^{3}/y Hw
375 N264-8 N264-8/dl-49, y Hw m^{2} g^{4}
379 N264-23 (mottled rst, fa; Tl;3L) y N264-23/dl-49, y Hw m^{2} g^{4}
380 N264-29 (mottled rst, fa dm; Tl;3L) y N264-29/dl-49, y Hw m^{2} g^{4}
383 N264-32a y N264-32/dl-49, y Hw m^{2} g^{4}

386 N264-34a (Tl; 3L) y N264-34/dl-49, y Hw m^2 g^4
388 N264-39 w^{ch} N264-39/dl-49, y Hw m^2 g^4
389 N264-40 N264-40/dl-49, y Hw m^2 g^4
390 N264-41 w N264-41/dl-49, y Hw m^2 g^4
391 N264-47 y N264-47/dl-49, y Hw m^2 g^4
392 N264-48 (In) y N264-48/dl-49, y Hw m^2 g^4
394 N264-50 (Tp 1 into 2L) y N264-50/dl-49, y Hw m^2 g^4
396 N264-53 (Tl;2L) y N264-53/dl-49, y Hw m^2 g^4
397 N264-54 y N264-54/dl-49, y Hw m^2 g^4
399 N264-57 (In) y N264-57/dl-49, y Hw m^2 g^4
400 N264-58b y N264-58/dl-49, y Hw m^2 g^4
401 264-58a-10 w spl, Dp
402 N264-59 (mottled w rst, fa; Tl;2L) y N264-59/w
403 N264-63 (Tp) y N264-63/dl-49, y Hw m^2 g^4
404 N264-64 (mottled w; Tl;3L) y N264-64/w
406 N264-66 (mottled w; Tpl;2R) y N264-66/w
407 N264-70 (mottled w rst, fa dm; complex Tl;3L;3R) y N264-70/dl-49, y Hw m^2 g^4
410 N264-72 (Df) y N264-72/dl-49, y Hw m^2 g^4
415 N264-83 (Complex T, In 1-3L) y N264-83/dl-49, y Hw m^2 g^4
416 N264-84 (mottled fa, dm; In) y N264-84/dl-49, y Hw m^2 g^4
418 N264-87a (Complex Tl;3R;2R) N264-87/dl-49, y Hw m^2 g^4
420 N264-88 (Df) N264-88/dl-49, y Hw m^2 g^4
424 N264-95 N/dl-49, Hw m^2 g^4
425 N264-97 y N/dl-49, Hw m^2 g^4
426 N264-100 (Tl;3L mottled w, spl, dm, ct) w sp N/y Hw w
428 N264-102 (Tl;2R) y N/dl-49, Hw m^2 g^4
429 N264-103 y N/dl-49, Hw m^2 g^4
431 N264-105 (Df N, dm) N/dl-49, Hw m^2 g^4
432 N264-107 y sc w N/dl-49, Hw m^2 g^4
433 N264-108 (In with Df rst, spl, dm) N/dl-49, Hw m^2 g^4
434 N264-109 N/dl-49, Hw m^2 g^4
435 N264-110 (Df w, rst, spl, dm) N/dl-49, Hw m^2 g^4
436 N264-111 (Df N) y N/dl-49, Hw m^2 g^4
437 N264-112 (In) y N/dl-49, Hw m^2 g^4
438 N264-114 (Df rst, spl, dm) N/dl-49, Hw m^2 g^4
440 N264-116 (In) N/dl-49, Hw m^2 g^4
441 N264-117 (Df w, rst, spl, dm) N/dl-49, Hw m^2 g^4
442 N264-118 (Df spl) N/dl-49, Hw m^2 g^4
443 N264-119 N/dl-49, Hw m^2 g^4
444 N264-120 (Df spl, dm) y N/dl-49, Hw m^2 g^4
446 N264-123 N/dl-49, Hw m^2 g^4
447 N264-126 (Df fa, dm) N/dl-49, Hw m^2 g^4
448 N264-128 N264-128/dl-49, Hw m^2 g^4
449 N264-129 N/dl-49, Hw m^2 g^4
450 N264-130 (Df fa) N/dl-49, Hw m^2 g^4
451 N264-132 y N/dl-49, Hw m^2 g^4
452 N264-133 (Complex Tl;2L;3R) N/dl-49, Hw m^2 g^4
453 N264-135 (N/dl-49), y Hw m^2 g^4 (Neel)
454 N264-136 y N264-136/dl-49, y Hw m^2 g^4
455 ct268-1a y ct268-1/dl-49, y Hw m^2 g^4
456 ct268-2a y ct268-a/dl-49, y Hw m^2 g^4
457 ct268-2b ct268-2/dl-49, y Hw m^2 g^4
458 ct268-3a y ct268-3/dl-49, y Hw m^2 g^4
459 ct268-5a (Tl;3R) ct268-5 B/dl-49, y Hw m^2 g^4
460 ct268-5b y ct^{-}B/dl-49, Hw m^2 g^4
461 ct268-6a ct268-6 sn^3/dl-49, y Hw m^2 g^4

462 ct268-17b (Tl;2R) y Hw ct268-17/sn^3
463 ct268-20 (In) y ct268-20/dl-49, y Hw m^2 g^4
464 ct268-21 (Tl;3R) y ct268-21 B/dl-49, y Hw m^2 g^4
465 ct268-23 y ct268-23 B/dl-49, y Hw m^2 g^4
466 ct268-24 (Tl;2R) y ct268-24/dl-49, y Hw m^2 g^4
467 st268-26 (Tl;2L) y ct268-26/dl-49, y Hw m^2 g^4
468 ct268-27 (In) y ct268-27/dl-49, y Hw m^2 g^4
469 ct268-29 (Tl;3R) y ct268-29/dl-49, y Hw m^2 g^4
470 ct268-29b y ct$^-$v f/dl-49, y Hw m^2 g^4
471 ct268-30 y ct268-30/dl-49, y Hw m^2 g^4
473 ct268-32 (Tl;2R & Tp) y ct268-32/dl-49, y Hw m^2 g^4
474 ct268-33 (Tl;2R) y ct268-33/dl-49, y Hw m^2 g^4
475 ct268-35 (?) y ct268-33/dl-49, y Hw m^2 g^4
476 ct268-36 (Tl;3L) y ct268-36/dl-49, y Hw m^2 g^4
477 ct268-37 (Tl;3L) ct$^-$/dl-49, Hw m^2 g^4
479 ct268-40 (complex Tl;2L;3L;3R) y ct$^-$/dl-49, Hw m^2 g^4
481 ct268-42 (Df) y ct$^-$/dl-49, Hw m^2 g^4
483 Df267-6 y v 267-6/dl-49, y Hw
484 Df259-4a (m) Df259-4/dl-49, y Hw m^2 g^4
485 Df259-4e (m) y Df259-4/dl-49, y Hw m^2 g^4
486 Df286-9 (dy) y Df286-9 B/dl-49, y Hw m^2 g^4
487 Df274-2a (Df wy Tl;2) sn Df274-2/dl-49, y Hw m^2 g^4
488 g271-2b (g-ty) y g271-2/dl-49, y Hw m^2 g^4
489 g271-3b (g-ty) (From Mrs. Morgan) y g271-3/dl-49, y Hw m^2 g^4
490 g271-6a (g ty) y g271-6/dl-49, y Hw m^2 g^4
491 g271-9 (g) y g271-9/dl-49, y Hw m^2 g^4
492 g271-10 (g) y g271-10/dl-49, y Hw m^2 g^4
493 f257-4 ++/y f
494 f257-5b (Tl;2) y f257-5/f B
495 f257-6a (From Pasadena) f257-6/f B
497 f257-19 f257-19 B/In AM
498 f257-22 (T) y f257-22 B^{i}B^i/In AM
499 f257-24 f257-24 B/In AM
504 Df B263-24a (In?) y Df B263-24/y f B
506 Df B263-34a Y B263-34/y f B
508 Df B263-43 y B263-43 (hom)
509 Df B263-46 y/B263-46
511 272-13 (complex Tp) y/dl-49, Hw m^2 g^4
512 278-2a y Bx$^-$/y f B
513 278-6 y B Bx$^-$/y
514 t282-1b y t$^-$/dl-49, Hw m^2 g^4
515 v267-4 (Tl;2L) y v B/dl-49, Hw m^2 g^4
516 285-2 (In) ec/dl-49, Hw m^2 g^4
517 285-3 ec/dl-49, Hw m^2 g^4
519 287-6a y/f B^1
520 pn323-1 (hom)
521 pn323-2 (hom)
523 pn323-7 (Dp) Dp +/y pn
524 pn323-8 (In) pn/dl-49, Hw m^2 g^4
525 337-2 rb sp/rb cx
526 251-33 N/dl-49, Hw m^2 g^4

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Wild Stocks

1 Canton-S
2 Lausanne-S
3 Oregon-R-C (Df(2)Ore)
4 Swedish-c (Df(2)Sw^{L}, Df(2)Sw^{R})
5 Urbana-S

Chromosome 1 (X)

6 amx/ClB
7 Ax
8 B
9 B^{36j}
10 Bg B/In(1)AM
11 bi ct^{6} g^{2}
12 bo
13 br
14 br w^{e} ec rb t^{4}/Ins(1)sc^{8}, dl-49, y^{31d} w^{a} lz^{s} B
15 Bx
16 Bx^{2}
17 Bx^{3}
18 Bx^{J}
19 Bx^{r}
20 car
21 ci-x y^{ci-x}
22 cm
23 cm ct^{6}
24 Co
25 ct^{n} oc/Ins(1)sc^{8}, dl-49, y^{31d} w^{a} lz^{s} B
26 cx
27 cx^{tg} t/Ins(1)sc^{8}, dl-49, y^{31d} w^{a} lz^{s} B
28 dd^{2}
29 dm/Cl, y Hw
30 dow/dl-49, m^{2} g^{4}
31 dwx/dl-49, m^{2} g^{4}
32 dy
33 ec
34 ec ct^{6} s car/Ins(1)sc^{8}, dl-49, y^{31d} w^{a} lz^{s} B
35 ec dx
36 ec dx/dl-49, Su-Hw y Hw m^{2} g^{4}
37 Ext/Ins(1)sc^{8}, dl-49, y^{31d} w^{a} lz^{s} B
38 f
39 f B
40 f BB/In(1)AM
41 f BB^{36b}/In(1)AM
42 f B^{3}/In(1)AM
43 f B^{i}B^{i}/yf:=
44 f fu/yf:=
45 f^{36a}
46 fa
47 flp
48 fo
49 fw^{35k}/y f:=
50 g^{2}
51 g^{2} pl/ClB
52 g^{2} ty/y f:=
53 gg^{2}/dl-49, y Hw w lz^{s}
54 gt bb^{l1}/ClB
55 gt w^{a}/ y f:=
56 gt w^{a}
57 Hw^{49c}/Muller-5
58 if^{3}
59 kz
60 l(1)7/dl-49, y Hw m^{2} g^{4}
61 lz/ClB
62 lz^{3}/y f:=
63 lz^{34k}/y f:=
64 lz^{37h}
65 m
66 ma-l/y f:=
67 M(1)o f/In(1)AM
68 M(1)Sp/In(1)AM
69 na/sc^{8}, dl-49, y^{31d} w^{a} lz^{s} B
70 ny
71 oc ptg^{3}/ClB
72 od
73 pn^{2}
74 ptg^{2}
75 r^{9}/In(1)AM ptg^{4}
76 r^{39k} f B/In(1)AM
77 ras dy
78 ras^{2}
79 ras^{3} m
80 rb
81 rb cx
82 rg
83 rst^{2}/dl-49, m^{2} g^{4}
84 s
85 sc
86 sc cv v f
87 sc ec cv ct^{6} v g f/ClB
88 sc^{2} pn/y f:=
89 sc^{3B}
90 sc$^{3-1}$ w
91 sc^{5} bb^{sc5}
92 sc^{6} w^{a}
93 sc^{10} w^{a}
94 sc$^{10-1}$/y Hw
95 scp t
96 Sh^{2}/Muller-5
97 shf^{2}
98 sn^{3}
99 sn^{4}
100 sn^{34e}
101 sn^{36a}/y f:=
102 spl
103 sta/ClB
104 Su^{x}-dx dx
105 su^{2}-s v
106 su^{2}-s w^{a} cv t f
107 su^{3}-s eq/y f:=
108 su^{S2}-v-pr v/y f:=
109 svr
110 svr w^{a}
111 sw
112 sx vb^{2} sy/In(1)AM
113 sy
114 t
115 t^{2} v f
116 t^{3}
117 tw/y Hw
118 un Bx^{2}/In(1)AM, ptg^{4}
119 un^{4}
120 v
121 v Bx^{r} car
122 v f su^{w}-f
123 v M(1)n/In(1)AM
124 v r^{l2}
125 v^{2} fw
126 v^{36f}
127 vb
128 vs
129 w
130 w m f
131 w^{a}
132 w^{a2}
133 w^{a3}
134 w^{a4}
135 w^{bf} f^{5}
136 w^{bf2}
137 w^{co} sn^{2}
138 w^{col}
139 w^{e}
140 w^{e2}
141 w^{h}
142 w^{i} f^{3} bb^{M}
143 w^{sat}

144 w^t fw
145 wy
146 y
147 y ac v
148 y pn
149 y ac
150 y sc lz^g v f/y f:=
151 y sc^5
152 y sc^{D1}
153 y sc^{D2}
154 y w spl
155 y^2
156 y^2 cv v f
157 y^2 dvr^2 v
158 y^2 w^a
159 y^2 wy^2 g^2
160 y^{2s}
161 y^{2s} fw^{34e}
162 y^{3d}/y f:=
163 y^{34c}
164 y^{td}
165 y^{v2}

Chromosome 2

166 a px sp
167 ab
168 ab^2/T(Y;2)E
169 abr/Cy, hk^2
170 ad
171 al
172 al b c sp^2
173 al dp b bw $\underline{l}$(2)ax/Cy, sp^2
174 al dp b pr blt bw/Cy, al^2 lt^3 L^4 sp^2
175 al dp b pr c px sp
176 dp^{o2}/T(Y;2;3)D; M(2)D
177 al S ast ho/Cy, E-S
178 Alu
179 an/Cy
180 an^2/Cy, bw^{v34}
181 ap^4/Rvd
182 arch chl/Cy, al^2 lt^3 L^4 sp^2
183 ast ho
184 ast^4 dp cl
185 b
186 b el rd^s pr cn
187 b Go/Cy, pr
188 b gp
189 b j
190 b $\underline{l}$(2)Bld pr c px sp/Cy, al^2 lt^3 L^4 sp^2
191 b lt bw/T(Y;2)G
192 b pr tk/T(Y;2)G
193 b rh/T(Y;2)H
194 b sf
195 b vg
196 bat/Ins(2L+2R)Cy, S^2
197 Bl/In(2LR)dp
198 Bl L^2/Cy sp^2
199 Bl stw^3/In(2LR)dp
200 Bl stw^{48} blt tuf/Cy, sp
201 Bla/Cy
202 blo
203 blt
204 bri
205 bs^2
206 bw
207 bw ba
208 bw tu
209 bw^{2b}
210 bw^D
211 c
212 c wt px
213 cg c/U
214 ch
215 chl
216 chl en/Cy, al^2 L^4 sp^2
217 chl $\underline{l}$(2)bw bw^{2b} mr^2/Cy, al^2 L^4 sp^2
218 chy
219 ck/Cy
220 cl
221 cl^2 px sp/T(T;2)E
222 cn
223 cn bw
224 cn en/Cy, al^2 lt^3 L^4 sp^2
225 cn^3/T(Y;2)C
226 cn^{35k}
227 cru/Cy; (w^e)
228 d/Cy(2L), dp^2 b pr
229 d b/Cy, pr
230 dil^2 hv bw sp/Cy, al^2 lt^3 L^4 sp^2
231 dke c
232 dp
233 dp^{Nov}
234 dp^o
235 dp^{o2}
236 dp^{Rf}/Pm, ds^{33k}
237 dp^t/Ins(2L;2R)Cy S^2 E-S
238 dp^{tx} b/Cy
239 dp^{v2}
240 dp^{v1}/Cy, al^2 L^4 sp^2
241 ds dp
242 ds ft dp^{v2} $\underline{l}$(2)M b pr/Cy dp^2
243 ds S G b pr/Cy, al^2 lt^3 L^4 sp^2
244 ds^W/In(2L)Cyt, Su-S dp^2 pr
245 ds^{38k}/Cy(2L), dp^2 b pr
246 dsr
247 dw-24F cl/Cy, dp^2
248 dw-24F l(2)cg cg/Cy, al^2 lt^3 L^4 sp^2
249 ed Su^2-dx
250 el
251 esc/Bl
252 ex
253 fes Alu lt/Cy, al^2 lt^3 L^4 sp^2
254 fj $\underline{l}$(2)Su-H/Xa
255 fj wt/Xa
256 fr/Cy, dp^2
257 fr^2 wt/Cy
258 ft
259 G^{rv}/Cy, dp^2
261 hk
262 hk pr
263 ho
264 hv/Cy, al^2 lt^3 L^4 sp^2
265 hy/Cy L^4 sp^2
266 hy a px sp/T(2;3)S^M, Cy
267 j
268 J Bl/In(2L)NS
269 J^{34e}
270 Jag
271 kn
272 L
273 L^2
274 L^4
275 L^5
276 Lr
277 L^{si}
278 $\underline{l}$(2)39a px slt sp/Cy, al^2 lt^3 L^4 sp^2
279 $\underline{l}$(2)36e/Cy; (hom.3)
280 $\overline{l}$(2) a bs^3, In(2L)t/Pm ds^{33k}
281 $\underline{l}$(2) ay b c sp/Cy, al^2 lt^3 L^4 sp^2
282 $\underline{l}$(2)H L^2/T(2;3)Xa
283 $\overline{l}$(2)mat/Cy
284 $\overline{l}l^2$
285 lm/Cy, S^2 dp^2 E-S
286 lt/T(Y;2)A
287 lt std/Cy, sp^2

288 lt stw^3
289 ltd
290 lw
291 M(2)173/Cy, al^2 lt^3 L^4 sp^2
292 M(2)B/In(2L)t, l(2)B
293 M(2)l^2/Cy, L^4 sp^2
294 M(2)p/Cy, al^2 lt^3 L^4 sp^2
295 M(2)S1/Cy, pr
296 M(2)S3/Cy, sp^2
297 M(2)S5/Cy, L^4 sp^2
298 M(2)S6/Cy, pr
299 M(2)S7/Cy, al^2 lt^3 L^4 sp^2
300 M(2)S9/Cy, dp^2
301 M(2)S11/Cy, bw^{V34}
302 M(2)S13/Cy, bw^{V34}
303 M(2)z/In(2L)t, l(2) R
304 M(2)z Sk b/Cy(2L), dp^2 b pr
305 mi/Pm^2
306 mr bs^2/Pm, ds^{33k}
307 mr^2/Bld, In(2R)Cy
308 msf/Cy, sp^2
309 net
310 net ed Su^2-dx
311 nw^2/Cy-RNS
312 pd
313 pd ll
314 pd ll^2 sp
315 Pfd/Ins(2L+2R)Cy, S^2
316 pi/Gla
317 pi l(2)30l/Cy, al^2 lt^3 L^4 sp^2
318 pk cn
319 pk tuf (sp^2/+)
320 po vg
321 po^2
322 pr
323 pr cn/T(Y;2)C
324 pr cn ix/Cy, al^2 lt^3 L^4 sp^2
325 pr^{bw}
326 pu
327 puff
328 pw-c/Cy
329 px
330 px bw sp/T(Y;2)J
331 px bw mr sp/Pm, ds^{33k}
332 pys
333 Q
334 rd/Cy, L^4 sp^2
335 rdo
336 rdo^2 pr
337 Rf/Pm, ds^{33k}
338 rh
339 rl
340 rub
341 S/Cy, E-S
342 S Sp ab^2 ltd/NS, px sp
343 S Sp Bl N-2/Cy, L^4 sp^2
344 S^R/Pm, ds^{33k}
345 sca
346 sca l(2)C/Cy, sp^2
347 Scd/Cy
348 sf^2
349 shr bw^{2b} abb sp/Cy, sp^2
350 shv
351 shv ho
352 Sk b/In(2L)NS
353 sm px/Cy
354 sm px pd/Cy, al^2 L^4 sp^2
355 sp^2 bs^2
356 Sp/In(2L)t, l(2)R
357 Sp J/In(2L)Cyt, Su-S dp^2 pr
358 Sp J L^2 Pin/Cy, sp^2
359 spd gt-4/Gla
360 std/Cy, al^2 lt^3 L^4 sp^2
361 stw/T(Y;2;3)I
362 stw^2
363 stw^3/T(Y;2)B
364 stw^5
365 stw^{48} blt tuf
366 Su-H whd l(2)Su-H/Cy, al^2 lt^3 L^4 sp^2
367 Su-H/Cy, pr
368 tkd/Cy, al^2 L^4 sp^2
369 tkv
370 tri vg^{No2}/Cy
371 tuf ltd
372 Uf
373 vg
374 vg l(2)C/Cy, sp^2
375 vg^{nl}
376 vg^{np}
377 vg^{nw} Hia/T(2;3)S^M Cy
378 vst/Cy
379 whd
380 wt

Chromosome 3

381 a-3
382 aa h
383 aa tu-36e
384 abd
385 app
386 as^{hg}
387 as^{hg} e^s
388 bar-3 (Ives)
389 Bd^G/In(3R)C, l(3)a
390 bf/In(3R)C, Sb e l(3)e
391 bul
392 bv
393 bx^3/In(3R)C, l(3)a (su^2-Hw present)
394 bx^{34e}
395 bx^D e^4/Payne, Dfd Ca
396 ca
397 ca bv
398 ca^2
399 cd
400 cmp ca/In(3R)C, e
401 cp
402 cp in ri p^p
403 cu
404 cu kar
405 cur
406 cv-c
407 cv-c sbd^2
408 cv-d
409 D/Gl
410 D^3 Sb ca^2/Payne
411 det
412 Dfd/Cx
413 Dfd^r
414 Dl H e^s cd/In(3R)spr, spr
415 Dl^3/In(3R)C, e
416 Dl^5/In(3R)C, l(3)a
417 Dl^7/In(3R)Mo, Sb sr
418 Dl^9/In(3R)C, e
419 Dl^{11}/Payne, Dfd ca
420 Dl^{12}/Payne, Dfd ca
421 Dl^{13}/In(3R)C, Sb e l(3)e
422 Dl^{14}/In(3R)Cyd, Cyd
423 Dl^x/Payne
424 dwh/Payne, Dfd ca
425 e^4 wo ro
426 e^{11}
427 e^s

428 e^{s} cd ro cmp ca/Xa,
ca
429 eg/Cx, D
430 eg^{2}/Cx, D
431 eyg
432 fz
433 gl
434 gl^{2} e^{4}
435 gl^{3}
436 Gl Sb/LVM
437 h
438 h cu H^{2} ca/Payne,
Dfd ca (l(3)Dl)
439 h^{2}
440 H/In(3R)hp, hp
441 H Pr/In(3R)C, e
442 H^{2}/Xa
443 H^{3}/In(3R)C, Sb e
l(3)e
444 Hn^{r}
445 in
446 jv
447 jv Hn^{r} h
448 jvl
449 kar^{2}
450 l(3)36d10/Cx D
451 l(3)ac e^{s} M(3)w/LVM
452 ld
453 Ly/D^{3}
454 Ly Sb/LVM
455 M(3)/In(3R)C,
e l(3)e
456 M(3)124/In(3R)C,
e l(3)e
457 M(3)36e/In(3R)C,
l(3)a
458 M(3)39b/In(3R)Cyd,
Cyd
459 M(3)40l30/Payne,
Dfd ca
460 M(3)B/In(3R)C,
e l(3)e
461 M(3)B^{2}/In(3R)C,
Sb e l(3)e
462 M(3)H/In(3R)Mo,
Sb sr
463 M(3)S32/T(2;3)Mé
464 M(3)S34/T(2;3)Mé
465 M(3)S36/T(2;3)Mé
466 M(3)S37/In(3L)Mé
467 M(3)w/In(3R)C,
e l(3)e
468 M(3)y/Mé
469 ma
470 ma fl
471 mah
472 Mc/Xa
473 obt
474 p
475 p^{p}
476 p^{p} bx sr e^{s}
477 p^{p} cu
478 pb/Cx, D
479 Pc/Mé
480 Pr/In(3R)C, e
481 Pt/Xa, ca
482 pyd
483 R Ly/In(3L)P, gm
484 ra
485 ri
486 ri bod e^{s}/Mé,
In(3R)C, Sb e
l(3)e
487 ri p^{p}/st, T(Y;2;3)F
488 ro
489 ro Bd ca/In(3R)C,
l(3)a
490 ro ca ra/T(2;3)Mé
491 rs^{2}
492 rsd
493 ru
494 ru h th st p^{p} H e^{s}
ro/C(3)C, M(3)X e^{x}
495 ru h th st cu sr e^{s}
ca (ru-reverted?)
496 ru h th st cu sr e^{s}
Pr ca/T(2;3)Mé
497 ru h th st p^{p} cu sr
e^{s}
498 ru^{g} jv se by
499 ry
500 Sb/In(3LR)bxd^{D101}
501 Sb bx^{D}/Xa
502 Sb H/In(3R)C, cd
503 Sb^{Spi}/Cx D
504 se
505 se app
506 se h
507 se rt^{2} th/Mé
508 se ss k e^{s} ro
509 sed
510 Ser/In(3R)C, e l(3)e
511 snb
512 sr
513 sr gl
514 sr sed
515 ss
516 ss bx Su^{2}-ss
517 ss bxd k e^{s}/Xa
518 ss^{a}
519 ss^{a-B}
520 st
521 st c3G ca/In(3)TM
(sp^{2})
522 st ri in p^{p}
523 st Sb^{r} e^{s} ro ca
524 st sr e^{s} ro ca;
tu 36-a
525 st sr H^{2} ca/In(3R)P^{W},
st l(3)W ca
526 st sp
527 su^{2}-Hw bx bxd/In-TM
528 su-t; t
529 su-ve ru ve h th
530 th
531 th st cp
532 th st pb p^{p}/Cx, D
533 tra/Cx, D (y v/w^{a})
534 tt wo
535 tu-h
536 tx
537 ve
538 ve h th
539 ve R/In(3L)P, gm
540 W
541 W Sb/Cx, D
542 wk/Payne Dfd ca
543 wo

Chromosome 4

544 ar/ey^{D}
545 bt
546 bt^{D}/ci^{D}
547 bt ey^{R} sv^{n}
548 ci ey^{R}
549 ci gvl bt
550 ci gvl ey^{R} sv^{n}
551 ci^{561}
552 ci^{D}/ey^{D}
553 ci^{W}
554 ci sv^{n}; ±♀; w^{a}♂
555 ey
556 ey^{2}
557 ey^{4}
558 gvl
559 gvl ey^{R}
560 gvl sv^{n}
561 gvl sv^{n} ey^{R}
562 Scn/ey^{D}
563 spa
564 spa^{Cat}/ci^{D}
565 sv^{35a}
566 sv^{de}/ey^{D}
567 sv^{n}

Multichromosomal Stocks

568 br^{3} dxst; ed Su^{2}-dx (1;2)
569 dxst; Su-dx (1;2)
570 e^{X}-S; S/Cy (1;2)
571 lz^{D}/Hw; Cy/Pm (1;2)
572 v; bw (1;2)
573 sy; tet (1;2)
574 ptg; px pd; su-pd (1;2;3)
575 al dp b Bl c px sp/Cy; D/C(3)X (2;3)
576 yf:=; bw; e; ci ey^{R} (1;2;3;4)
577 bw; st (2;3)
578 Cy/Pm ds^{33k}; H/In(3R)Mo, Sb sr (2;3)
579 dp^{v}; vo^{3} (2;3)
580 Pm, dp b/Cy, sp^{2}; Sb/D, CxF (2;3)
581 px pd; Pdr H Dp(2;3)P/Pdr (2;3)
582 l(2)gl; l(3)tr/T(2;3)E (2;3)
583 Mal pr

Attached-X

584 +/sc^{2} pn
585 br ec/y^{3d}
586 f B/su^{S2}-v-pr v
587 w^{bf} M(1)36/w^{bf3}/sn^{36a}
588 y/g^{2} ty
589 y v .... (533)
590 y v f .... (600)
591 y w .... (763)
592 y w bb/f B^{i}B^{i}
593 y v f car .... (599)
594 y^{2} v f car .... (671)
595 y^{2} su-w^{a} w^{a} bb .... (703)

Closed-X

596 X^{c2}
597 X^{c2}, cv v f/ClB
598 In(X^{c2})w^{vc}/y Hw dl-49 m^{2} g^{4} f^{5} (Catcheside)

Triploid (3N)

599 sc^{S1} B In-S w^{a} sc^{8}/y v f car

Closed-Y

600 Y^{lc}/y w Y^{S} & y v f (Muller)

Deficiencies

Deficiencies-X

601 Df(1)259-4	Df(1)259-4/dl-49 y Hw m^{2} g^{4}
602 Df(1)(O+ac)260-2	(647, 649, etc.)
603 Df(1)(O+sc)260-1	Df(1)(O+sc)/dl-49, y Hw m^{2} g^{4}
604 Df(1)B263-20	Df(1)B263-20/sc^{7} AM
605 Df(1)bb	In(1)bb-, y sl^{2} bb-/In(1)AM (Dobzhansky)
606 Df(1)bb	In(1)bb-, y v car bb-/In(1)AM
607 Df(1)bb$^{\underline{l}}$	(621)
608 Df(1)ct268-30	Df(1)ct268-30, y/dl-49, y Hw m^{2} g^{4}
609 Df(1)ct268-42	Df(1)ct268-42, y/dl-49, y Hw m^{2} g^{4}
610 Df(1)g^{l}	Df(1)g$^{\underline{l}}$, f B/In(1)AM (L. V. Morgan)
611 Df(1)N8	Df(1)N8/dl-49, y Hw m^{2} g^{4} (Mohr)
612 Df(1)N264-39	Df(1)N264-39 w^{ch}/dl-49, y Hw m^{2} g^{4}
613 Df(1)N264-105	Df(1)N264-105(dm)/dl-49, y Hw m^{2} g^{4}
614 Df(1)rst^{2}	(83)
615 Df(1)svr	Df(1)svr, Dp(1;f)101 (Dp. het or hom.)
616 Df(1)t282-1	Df(1)t282-1, y t-/dl-49, y Hw m^{2} g^{4}
617 Df(1)w258-45	Df(1)w258-45, y/dl-49, y Hw m^{2} g^{4}
618 Df(1)w258-48	Df(1)w258-48/dl-49, y Hw m^{2} g^{4}
619 Df(Y)Y^{bb-}	y^{2} eq; Df(Y)Y^{bb-}/y w bb Df(Y)Y^{bb-}
620 Df(Y)Y^{bb-rev}	Y^{lS} su^{3}-s eq; Y^{bb-rev}/y w bb Y^{bb-rev}
621 Df(Y)st	w^{e} bb$^{\underline{l}}$/w^{e} bb$^{\underline{l}}$; Yst & w^{e} bb$^{\underline{l}}$; Y +; NS, px sp/l mr^{2} (Bridges)
622 Df(Y)Y"	XY', g^{2} B; Y"/y; Y(Het. In(2R)Cy) (Stern)

Deficiencies-2

623 Df(2)42	Df(2)42, en/Cy, al^{2} lt^{3} L^{4} sp^{2}
624 Df(2)al	Df(2)al/Cy, E-S
625 Df(2)bw^{5}	Df(2)bw^{5} sp^{2}/Xa
626 Df(2)bw^{VDe2}	Df(2)bw^{VDe2} Cy^{R}/Gla
627 Df(2)M33a	Df(2)M33a/Pm^{2}
628 Df(2)MB	Df(2)MB/Cy, al^{2} lt^{3} L^{4} sp^{2}
629 Df(2)M1	Df(2)M1/Cy, sp^{2}
630 Df(2)MS2	Df(2)MS2/Cy, pr
631 Df(2)MS4	Df(2)MS4/Cy, L^{4} sp^{2}
632 Df(2)MS8	Df(2)MS8/Cy, al^{2} lt^{3} L^{4} sp^{2}
633 Df(2)MS10	Df(2)MS10/Cy, pr
634 Df(2)Px	Df(2)Px/Df(2)P; Df(2;3)P/In(3R)Mo, Sb sr; w^{e}
635 Df(2)Px^{2}	Df(2)Px^{2}, bw sp/Ins(2L+2R) Cy, S^{2} E-S
636 Df(2)rl^{10a}	Df(2)rl^{10a} lt cn/Cy
637 Df(2)rl^{43}	Df(2)rl^{43} lt cn/Cy
638 Df(2)S^{2}	Df(2)S^{2}/Cy E-S
639 Df(2)S^{3}	Df(2)S^{3}/Dp(2;2)A, Cy, E-S
640 Df(2)vg^{B}	Df(2)vg^{B}/Cy, L^{4} sp^{2}
641 Df(2)vg^{C}	Df(2)vg^{C}/Rvd
642 Df(2)vg^{S}	Df(2)vg^{S}, Cn/Cy, al^{2} lt^{3} L^{4} sp^{2}

Deficiencies-3

643 Df(3)Ly	(453, 454)
644 Df(3)MS31	Df(3)MS31/T(2;3)Me
645 Df(3)sbd^{105}	Df(3)sbd^{105}/Xa

Deficiencies-4

646 Df(4)M4	Df(4)M4/ey^{D}

Duplications

647 Dp(1;f)101	In(1)sc^{8}, Df(0+ac) w^{a} sc^{8} Dp(1;f)101
648 Dp(1;f)101	(615)
649 Dp(1;f)107	In(1)sc^{8}, Df(0+ac) w^{a} sc^{8}; Dp(1;f)107
650 Dp(1;f)118	In(1)sc^{8}, Df(0+ac) w^{a} sc^{8}; Dp(1;f)118
651 Dp(1;f)135	In(1)sc^{8}, Df(0+ac) w^{a} sc^{8}; Dp(1;f)135 y^{2}
652 Dp(1;f)X^{c2}	Dp(1;f)X^{c2} y l(1)7/y l(1)7
653 Dp(1;1)112	y f, Dp(1;1)112 (homozygous stock)
654 Dp(1;YL)sc^{S1}	sc^{S1}.Y^{L}/y.Y^{S}; y f:=; cn bw (e)
655 Dp(1;Y)sc^{8}	sc^{8}.Y/X^{c2}, t^{2}; cn bw (Muller)
656 Dp(1;3)126	v f; Dp(1;3)126/Payne Dfd ca
657 Dp(Y;1)Su-4	B bb, Dp(Y;1)Su-4 (Stern)
658 Dp(2;2)S	Dp(2;2)S (+ast) (+ast) ho (hom.)
659 Dp(2;2)S	Dp(2;2)s (S ast) (S ast^{4}) net dp cl/Cy, E-S
660 Qn(2;2)S	Qn(2;2)S, (+ast)^{5}, al ho/Cy, S^{2} E-S

Inversions

Inversions-X

661 In(1)AB	In(1)AB/y v f
662 In(1)AM	(10, 40, etc.)
663 In(1)B^{M1}	In(1)B^{M1} v (tan-like)
664 In(1)B^{M2}	In(1)B^{M2} v^{+} (rev)
665 In(1)B^{M2}	In(1)B^{M2} (rev) f^{B} l 5 (rein.; mosaic)

666	In(1)B^{M2}	In(1)B^{M2} f^{B27}/ClB (mosaic in f/f^{27})
667	In(1)bb-	(605, 606)
668	In(1)Cl	(In ClB in 6, 51, etc.; with y Hw in 29)
669	In(1)dl-49	dl-49, cm^{2}
670	In(1)dl-49	dl-49, ty-1
671	In(1)dl-49	dl-49, ty-1 bb^{1}/y^{2} v f car
672	In(1)dl-49	dl-49, v^{0} f
673	In(1)dl-49	dl-49, y fa^{n}
674	In(1)dl-49	(y Hw m^{2} g^{4} in 60, etc.; y Hw w lz^{s} in 53)
675	In(1)dl-49, B^{M1}	In(1)dl-49, sc v B^{M1} (homozygous)
676	In(1)rst^{3}	In(1)rst^{3}, rst^{3} (homozygous)
677	In(1)rst^{3}	In(1)rst^{3}, y rst^{3} car bb
678	In(1)sc^{4}	In(1)sc^{4}, y sc^{4}
679	In(1)sc^{7}	In(1)sc^{7}, sc^{7}
680	In(1)sc^{7}	In(1)sc^{7}, sc^{7}, w^{a}
681	In(1)sc^{7}	In(1)sc^{7}, sc^{7} w^{a} fa^{2} sn^{3} v
682	In(1)sc^{7}, AM	(604)
683	In(1)sc^{8}	In(1)sc^{8}, sc^{8}
684	In(1)sc^{8}	In(1)sc^{8}, sc^{8} cv v f/y f:=
685	In(1)sc^{8}	In(1)sc^{8}, y^{31d} sc^{8} w^{a}
686	In(1)sc^{3}	sc^{8} B In-S w^{a}
687	In(1)sc^{8}, dl-49	(with y^{31d} w^{a} lz^{s} B in 14, 25, etc.)
688	In(1)sc^{9}	In(1)sc^{9}, sc^{9} Bx f t w^{a}/w dl-49 lz^{s}
689	In(1)sc^{J1}	In(1)sc^{J1}; Del 24
690	In(1)sc$^{260-14}$	In(1)sc$^{260-14}$, sc$^{260-14}$
691	In(1)sc$^{260-22}$	In(1)sc$^{260-22}$, sc$^{260-22}$

Scute combinations isogenic for X (Raffel and Muller)

692	In(1)sc^{L8}	In(1)sc^{L8}, sc^{L8} car m w^{a}/y dl-49 lz^{s}
693	In(1)sc^{L8} sc^{S1}	In(1)sc^{L8}, sc^{L8} car m w^{a}, In(1)sc^{S1}, sc^{S1}/ y w dl-49 lz^{s}
694	In(1)sc^{S1}, sc^{8}	In(1)sc^{S1}, sc^{S1} car m w^{a}, In(1)sc^{8}, sc^{8}/w dl-49 lz^{s}
695	In(1)sc^{S1}, sc^{L8}	In(1)sc^{S1}, sc^{S1} L car m w^{a}, In(1)sc^{L8} sc^{L8}/ y w dl-49 lz^{s}
696	In(1)sc^{4}	In(1)sc^{4}, y sc^{4} car m w^{a}/w dl-49 lz^{s}
697	In(1)sc^{4}, sc^{8}	y In(1)sc^{4}, sc^{4} car m w^{a}, In(1)sc^{8}, sc^{8}/ y w dl-49 lz^{s}
698	In(1)sc^{4}, sc^{S1}	y In(1)sc^{4}, sc^{4} car m w^{a}, In(1)sc^{S1}, sc^{S1}/ w dl-49 lz^{s}

Remaining X-chromosome inversion stocks

699	In(1)w^{m4}	In(1)w^{m4} (bb?)
700	In(1)w^{m4}	In(1)w^{m4}, y^{48h} w^{48h} sn^{48h} m^{48h} (Lindsley)
701	In(1)y^{4}	In(1)y^{4}, y^{4}
702	In(1)y^{4}	In(1)y^{4}, y^{4} cv v f
703	In(1)sc^{4}, sc^{8}	In(1)sc^{4}, sc^{8}, y/y^{2} su-w^{a} w^{a} bb

Inversions-2

704	In(2)bw^{VDe1}	In(2)bw^{VDe1}/b lt l cn mi sp
705	In(2)bw^{VDe2}	In(2)bw^{VDe2}/Rev. l

2L Inversions

706	In(2L)Cy	In(2L)Cy, al^{2} ast^{3} b pr (Cy not present)
707	In(2L)Cy	(with Cy dp^{2} b pr in 245, 304, etc.)
708	In(2L)Cyt	(with Su-S dp^{2} pr in 244, 357)
709	In(2L)ho	(793)

710 In(2L)NS	(268, 352)
711 In(2L)t	In(2L)t, lt l L^4 sp^2/Pm, ds^{33k}
712 In(2L)t	(with l(2)B in 292, with l(2)R in 303)

2L + 2R Inversions

713 Ins(2L+2R)Cy	(in balancers as Cy; Cy S^2 and Cy, S^2 E-S; S^2 and S^2 E-S do not carry Curly mutant)
714 Ins(2L+2R)Cy, (2R)bw^{V34}, Cy	(180, 301, 302)
715 Ins(2L)Cy, (2R)NS, Cy	(311)
716 Ins(2L+2R)NS	NS, mr/Cy
717 Ins(2L+2R)NS	(with px sp in 342, 621)

2LR Inversions

718 In(2LR)dp	(197, 199)
719 In(2LR)Gla	(316, 359)
720 In(2LR)Pm	(with ds^{33k} in 236, etc.)
721 In(2LR)Pm^2	(305, etc.)
722 In(2LR)Rvd	(181, 641)
723 In(2LR)U	(215)
724 In(2LR)Rev	(705)

2R Inversion

725 In(2R)Cy	(307)

Inversions-3

3L Inversions

726 In(3L)D	(D and D^3 stocks)
727 In(2L)$P^{mot-36e}$	In(3L)$P^{mot-36e}$/R
728 In(3L)P	(with gm in 483; with Mé in 507, etc.)

3L + 3R Inversions

729 Ins(3L+3R)P	$Payne^{ra}$, ra/ra l(3)ra
730 Ins(3L+3R)P	(as LVM, Payne; and Payne Dfd ca)

3LR Inversions

731 In(3LR)Cx, D	(429, 430, etc.)
732 In(3LR)sep	In(3LR)sep, sep ri p^P

3R Inversions

733 In(3R)C	(with cd in 502, with e in 441, with e l(3)e in 455, with Sb e l(3)e in 461)
734 In(3R)Dl^B	In(3R)Dl^B, st Dl^B/In(3R)P^W, st l(3)W ca
735 In(3R)Cyd	(422, 453)
736 In(3R)hp	(440)
737 In(3R)Hu	In(3R)Hu, Hu Sb^{Spi}/ Payne
738 In(3R)Mo	In(3R)Mo, sr/Xa, ca
739 In(3R)Mo	(with Sb sr in 417, 578, etc.)
740 In(3R)P	In(3R)P^{FLA} (homozygous)
741 In(3R)P	(with st l(3)W ca in 734)

Translocations

Translocations-1;2

742	T(1;2)106	T(1;2)106 (hom. ♀; het. ♂)
743	T(1;2)B^{bd}	T(1;2)B^{bd}/Cy M(2)e/Cy ♂
744	T(1;2)Bld	T(1;2)Bld, Bld/C1B (carries In(2R)Cy)
745	T(1;2)f257-5	T(1;2)f257-5/In(1)AM (Demerec)
746	T(1;2)lt	T(1;2)lt/Cy (carries eq. possibly su^{3}-s)
747	T(1;2)N264-9	T(1;2)N264-9/dl-49, w lz^{s} (=N^{9})
748	T(1;2)N264-10	T(1;2)N264-10/y w dm (=N^{10})
749	T(1;2)sc^{S2}	T(1;2)sc^{S2}/Cy
750	T(1;2)wy274-2	T(1;2)wy274-2, w^{a} sn B/In(1)AM

Translocations-1;3

751	T(1;3)1	T(1;3)1, In(3R)P Dfd ca/sc pn^{3} g^{2} f Bx^{2}
752	T(1;3)3	T(1;3)3 (hom. ♀; het. ♂)
753	T(1;3)263-4	T(1;3)263-4, y sc B^{1}/In(1)AM
754	T(1;3)283-3	T(1;3)283-3/w^{e} sn
755	T(1;3)"Del. 143"	T(1;3)"Del. 143"; ru 3L.XF; sc^{+}. 3R e^{s} ca/ ru h D CxF ca (D lost)
756	T(1;3)N264-6	T(1;3)N264-6 y/y w dm(=N^{6})
757	T(1;3)04	T(1;3)04/C1B
758	T(1;3)05	T(1;3)05 D/y
759	T(1;3)sc^{J4}	(1 y ac) sc^{8} w^{3}; T(1;3)sc^{J4} L.
760	T(1;3)sc260-15	T(1;3)sc260-15/dl-49, y Hw m^{2} g^{4}
761	T(1;3)v	T(1;3)v, sc cv v f/In(1)y^{4}, y^{4} sn w^{a}("hi-non")
762	T(1;3)w^{amK2}	T(1;3)w^{amK2}, sc^{8} B/w dl-49 lz^{s}

Translocations-1;4

763	T(1;4)A13	T(1;4)A13/y w
764	T(1;4)B^{S}	T(1;4)B^{S}/y f:=; bw; e; ci ey^{R}
765	T(1;4)N^{8a}	T(1;4)N^{8a}/dl-49 w lz^{s}
766	T(1;4)N264-84	T(1;4)N264-84, y/dl-49, y Hw m^{2} g^{4}
767	T(1;4)sc^{8}	T(1;4)sc^{8}, B w^{a}/y f :=
768	T(1;4)w^{m5}	T(1;4)w^{m5}
769	T(1;4)w^{m5}(1;3)sc^{J4}	T(1;4)w^{m5}; T(1;3)sc^{J4R} (C1B)
770	T(1;4)w^{VD3} (=w$^{258-21}$)	T(1;4)w^{VD3}/y w dm
771	T(1;4)w258-18	T(1;4)w258-18 y/y w dm

Translocations-Y;2

772	T(Y;2)A	(286)
773	T(Y;2)B	T(Y;2)B/b c
774	T(Y;2)C	(225, 323)
775	T(Y;2)E	(168, 221)
776	T(Y;2)G	(191, 192)
777	T(Y;2)H	(193)
778	T(Y;2)J	(330)
779	T(Y;2)rl	T(Y;2)rl, lt cn/b lt bw

Translocations-Y;2;3

780	T(Y;2;3)D	(176)
781	T(Y;2;3)F	(487)
782	T(Y;2;3)I	(361)

Translocations-2;3

783	T(2;3)101	al T(2;3)101 sp^2/Cy L^4 sp^2
784	T(2;3)101	ru h T(2;3)101 e^4 ro ca/Payne, Dfd ca
785	T(2;3)108	al T(2;3)108 c sp^2/Cy, al^2 lt^3 L^4 sp^2
786	T(2;3)109	T(2;3)109 p^P/Payne Dfd ca
787	T(2;3)A	Bl T(2;3)A; ru h D TA ss e^s/Payne
788	T(2;3)B	al Bl T(2;3)B sp^2/Cy, L^4 sp^2
789	T(2;3)B	T(2;3)B; ru h D TB ss e^s/Payne
790	T(2;3)bw^{VDe3}	T(2;3)bw^{VDe3}/Cy, ru h st ca
791	T(2;3)bw^{VDe4}	T(2;3)bw^{VDe4}/Cy, dp^2
792	T(2;3)C	Bl T(2;3)C; ru h D TC ss e^s/Payne
793	T(2;3)dp150	In(2L)ho T(2;3)dp150/Cy, E-S
794	T(2;3)E	T(2;3)E/Cy; D
795	T(2;3)Mé	(493, 494, 496)
796	T(2;3)P	w^e; Df(2)P/Cy; Dp(2;3)P
797	T(2;3)p^{Gr}	T(2;3)p^{Gr}/Cy
798	T(2;3)Dp-S	T(2;3)Dp-S; ho/Cy, E-S (hom. viable)
799	T(2;3)Xa	T(2;3)Xa/$\underline{1}$(3)Xa R
800	T(2;3)Xa	(in 501, 517, etc.; with ca in 428)

Translocations-2;4

801	T(2;4)a	T(2;4)a/Cy, pr; ey^2
802	T(2;4)ast^V	T(2;4)ast^V/Cy al^2 lt^3 L^4 sp^2
803	T(2;4)b	T(2;4)b/Cy, pr; ey^2
804	T(2;4)d	al dp T(2;4)d px sp/Cy pr; ey^2
805	T(2;4)d	T(2;4)d/Cy, pr

Translocations-3;4

806	T(3;4)a	D T(3;4)a/Mé
807	Y(3;4)c	T(3;4)c/Payne, Dfd ca
808	T(3;4)e	T(3;4)e, D/Mé
809	T(3;4)e	h th st T(3;4)e cu sr e^s ca/Payne, Dfd ca
810	T(3;4)f	T(3;4)f, h th st cu sr e^s ca/Payne, Dfd ca
811	T(3;4)f	T(3;4)f/Mé

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(This supersedes all previous lists)

a. Wild Stocks

No.	Genotype
a1	+ Amherst 3 (homoz. Singh, 1939)
a2	+ Crimea
a3	+ Florida 5 (homoz. Singh, 1939)
a4	+ Samarkand
a5	+ (iso 2, $a)
a6	+ (iso 2, $b)
a7	+ (iso 2, $c; may be ro-like in some 3rd chrom.)

b. Chromosome 1 (X)

b1	ac^3 (= sc^{10}) w^a
b2	ac^3 w^a & ac^3 w^a ct f.=
b3	ac^3 w^a.Dp sc^{V1} & y f:=

No.	Genotype
b4	At & y f:=
b5	B
b6	(B^{i}) f B^{i} (Luce 436.1)
b7	(B^{M1}-1) cm B^{M1},In & y f:=
b8	(B^{M1}-2) sn^{3} v B^{M1} & y f:=
b9	(B^{M1}-3) v m B^{M1},In & y f:=
b10	(B^{M1}-4) y rb cx B^{M1},In / C$\underline{l}$ B
b11	(B^{M2}) v^{+} (rev.v) B^{M2},In
b12	(B^{M2}rein.) v^{+}(rev.v) B^{M2+}(rev.B, rein.) f^{B15}(mosaic)
b13	(bb^{-},In-1) y sl^{2} bb^{-},In / InAM
b14	(bb^{-},In-2) y v car bb^{-},In / y InAM
b15	bo
b16	cm
b17	cm ct^{6} sn^{3} & y f:=
b18	ct^{n} oc / y^{31D} sc^{8} B In49 lz^{s} w^{a}
b19	ct^{6} v dy g f / InA99 sn^{33f}
b20	("doubler") y w^{a}.Dp(B^{S}) / sc^{S1} In49 v
b21	f B (Luce "L237B")
b22	f B B / In,AM
b23	f B od car / sc^{S1} f In49 v
b24	f fu / C$\underline{l}$ B
b25	f^{5} su-f
b26	$f^{ex18aH1}$ car / y sc^{S1} B InS
b27	f^{x} car & y f:=
b28	f^{x} Dp(f^{+ih})
b29	g^{2} pl & y f:=
b30	g^{2} ty & y f:=
b31	g^{im} sd^{s} / y sc^{S1} B InS
b32	g^{w}
b33	g^{x},Inh & y f:=
b34	In49 & y f.=
b35	In49 sn^{x2} & y f.=
b36	In49 v sn^{x2} B & y f:=
b37	("leftester 1") sc^{J1} pn w rb cm ct / y sc^{8} f In49 w^{a}
b38	("leftester 2") sc^{J1} oc ptg B^{M1} / y sc^{S1} f In49 v sc^{8}
b39	lz & y f:=
b40	("maple") y ac sc pn w rb cm ct^{6} ras^{2} v g^{2} f car & y f:=; sc^{19i} / Cy
b41	N^{8} / y Hw In49 m g
b42	oc ptg^{3} / C$\underline{l}$ B
b43	oc ptg Tu / sc^{S1} fu In49 sc^{8}
b44	od Dp(f^{+ih}) & y f:=
b45	or (overripe eye) & y f:=
b46	("plex-Tu") y ac sc pn w rb cm ct^{6} sn^{3} ras^{2} v dy g f Tu car / y sc^{S1} g In49 m sc^{8}
b47	pn,Inh $\underline{l}$ / y Hw In49 m g
b48	ras^{2}
b49	ras^{4} m / C$\underline{l}$ B
b50	rg^{7},In / sc^{S1} B InS
b51	rst^{3},In & y f:=
b52	rst^{-}(=rst^{2}) / y Hw In49 m g
b53	Sc (Scotched eye) / y Hw In49 m g
b54	sc ct^{6} car & y f:=
b55	sc Sc v f car / sc^{S1} B InS
b56	sc t^{2} v car & y f:=
b57	sc t^{2} v f Tu car & y f:=
b58	sc t^{2} v f & y f:=

No.	Genotype
b59	sc w BB^{L},In.Y^{s} & y f:=
b60	sc w (fB)$^{-L}$,In.Y^{s} / sc^{S1} f In49 v
b61	sd f & y f:=
b62	sd f / y sc^{8} B f In49 v
b63	sn^{3}.Dp(sc^{S1}) & y f:=
b64	sn^{3} lz^{46f24} ras^{4} v & y f:=
b65	sn^{c} / y Hw In49 m g
b66	spl rb cx & y f:=
b67	(tandem X.X) y^{2} X^{+}.sc^{8} w^{a} InS B ♀ & y^{S1} sc^{8} sn^{5} w ♂
b68	t^{2} & y f:=
b69	("tester 1") y ac pn w rb wy^{2} g^{2} & y f:=; sc^{19i} / Cy
b70	("tester 2") y^{2} w^{a} cm wy^{2} g^{2} car & y f:=; sc^{19i} / Cy
b71	("tester 3") y rb cm ras^{2} g^{2} & y f:=; sc^{19i} / Cy
b72	Tu & y f:=
b73	un Bx / InAM ptg^{4}
b74	w
b75	w$^{a'}$; (b cn cru mr / InsNS px sp)
b76	w^{e}
b77	w^{ec3} (ecru)
b78	w^{m} R7aH1
b79	w^{m4},In (3V1-2 & 20)
b80	w^{m4},In g? v w^{mMc} / y ac v
b81	w^{mMc},In & y f:=
b82	w^{mMc},In f w^{m4}
b83	(w^{r}-reddish) sc^{8} InS w^{r}
b84	(w^{r}-reddish-B) sc^{8} B InS w^{r} & y f:=
b85	Xc^{2} & y f:=
b86	Xc^{2} t^{2} & y f:=
b87	Xc^{2} v & y.= (bw; e; ey)
b88	Xc^{2} y v
b89	Xc^{3}(tm-ac) w^{a} InS B & y f:= / sc^{8}.Y (rings from tandem X.X)
b90	y ac dvr$^{(+)}$ v bb
b91	(y ac)$^{-}$ f (from y f:=) & y f:=
b92	y ac lz^{g} v f & y f:=
b93	y ac pn w rb cx (/ y sc^{4} B InS w^{a} sc^{8})
b94	y ac pn w rb cm ct^{6} sn^{3} oc ras^{2} v dy g f od car sw / y sc^{S1} B In49 v w^{a} sc^{8}
b95	y ac sc pn w.Dp sc^{V1} & y f:=
b96	y ac sc pn w rb cm ct^{6}; sc^{19i} / & y f:=
b97	y ac sc pn w spl rb cx. (sc^{19i}(b pr)/) & y f:= (sc^{19i}(b pr)/)
b98	y ac sc pn & y f:=
b99	y ac sc v & y f:=
b100	y ac t^{2}.Dp(y^{+}sc^{S1}) & y f:=
b101	y ct^{6} & y f:=
b102	y ct^{6} dvr^{2} v f & y f:=
b103	y ct^{6} f & ac w^{a} ct f.=
b104	y ct^{6} f car & y f:=
b105	y ct^{6} f.Dp(y^{+}sc^{V1}) & y f:=
b106	y ct^{6} lz.Y^{s} & y f:=
b107	y ct^{6} t^{2} v f car & y f:=
b108	y fa v & y f:=
b109	y fa wy^{2} g^{2}
b110	y In49 m f car & y f:=
b111	y In49 sn^{x2} B.Y^{l} & y f.=
b112	y In49 sn^{x2} bb^{l} & y f:=
b113	y In49 v B & y f:=

No.	Genotype
b114	y In49 y f car & y f:=
b115	y rst^{3},In car bb
b116	y sc
b117	y sc v g & y f:=
b118	y sn v & y f:=
b119	y t^{2} v f
b120	y v (isogenic)
b121	y v f^{x} Dp(f^{+ih}) & y f:=
b122	y w f
b123	y w f.Dp(sc^{S1})
b124	y w In49 f
b125	y w In49 lz^{s}.Dp(y sc^{V1}) & y f:=
b126	y w sn^{3}
b127	y w$^{-258-11}$ $\underline{l}$ / y Hw In49 m g
b128	y w t^{2} v f & y f:=
b129	y w$^{m258-18}$ t v f & y f:=
b130	y w$^{-258-11}$ t v f / y sc^{S1} B InS
b131	y w$^{m258-18}$ t^{2} v & y f:=
b132	y^{2} ec cv v f car
b133	y^{2} oc ptg B^{M2} & y f.=
b134	y^{2} oc ptg g,Inh & y f.=
b135	y^{2} v
b136	y^{2} v f car
b137	y^{2} v f car.Dp(y^{+}sc^{V1}) & y f:=
b138	y^{2} v f car su-f & y f:=
b139	y^{2} w^{a} ct f.Dp(sc^{S1}) / C$\underline{l}$ B
b140	y^{2} w^{a} ct mw f / y sc^{S1} B InS
b141	y^{2} w^{a} InS B
b142	y^{2} w^{a} sn^{5} B & y f:=
b143	y^{2} w^{a} v
b144	y^{3P},In B
b145	y^{4},In w^{a}
b146	y^{18cH1}

c. Scute alleles

(Listed alphabetically according to scutes, regardless of position of the scute in linear order)

c1	sc^{2} pn
c2	y sc^{4} B v^{41b}/ y w In49 lz^{s}
c3	y sc^{4} In49 w^{a} & y f:=; Cy / sc^{19i}
c4	y sc^{4} InS w^{a}; S sc^{19i} Bl / Cy L^{4} sp
c5	y sc^{5}
c6	sc^{6} car
c7	sc^{7} oc ptg g,Inh & y f:=
c8	sc^{7} w^{a}
c9	sc^{8} B
c10	sc^{8} B In49 & y f:=

Age Exp. struct. changes Lethals in sc^{8} B chromosome sc^{8} B $\underline{l}$ w^{a} / w sn ♀ & w sn ♂ (c11-c16)

c11	♀$\underline{l}$ 18 Allay (Dob.6)
c12	♀$\underline{l}$ 18 Al2ay (Dob.5)
c13	♀$\underline{l}$ 23 Bl8io (Dob.2)
c14	♀$\underline{l}$ 53 Bl4d,am (A7)
c15	♀$\underline{l}$ 69 Bl4ay (nd, c.o.)
c16	♀$\underline{l}$ 74 Allam Notch
c17	sc^{8} B In49 m & y f:=

No.	Genotype
c18	sc^{8} bb w^{a}
c19	sc^{8} car f In49 v & y f:=
c20	sc^{8} f dl49 v & y f:=
c21	sc^{8} g In49 & y f:=
c22	sc^{8} g In49 m / w sn^{5s}
c23	sc^{8} In49 v
y^{-} from X-raying oögonia	y^{-} sc^{8} B / y Hw In49 m g ♀ & y Hw In49 m g ♂ (c24-c28)
c24	y♀3$\underline{1}$ A2
c25	y♀3$\underline{1}$ A16
c26	y♀3$\underline{1}$ C2
c27	y♀3$\underline{1}$ C10
c28	y♀3$\underline{1}$ C21
c29	(y ac)^{B270} (dappled) sc^{8} B w^{a} / w In49 lz^{s}
c30	y^{S1} sc^{8}
c31	y^{S1} sc^{8} B f In49 v w^{a} & y f:=
c32	y^{S1} sc^{8} B In49
c33	y^{S1} sc^{8} B In49 lz^{s} w^{a} / na
c34	y^{S1} sc^{8} B f In49 v & y f:=
c35	y^{S1} sc^{8} f In49 lz^{s} / w sn^{5s}
c36	y^{S1} sc^{8} f InS w^{a}
c37	y^{S1} sc^{8} In49 v m & y f:=
c38	y^{S1} sc^{8} sn^{3} w
c39	y^{OX} sc^{8} sn^{5} w & y f:=
c40	sc$^{3.1}$ / y Hw
c41	sc^{19-}; fes sc^{19i} b pr / Cy dpTh pr
c42	sc^{19-}♂ & y f:=♀; fes sc^{19i} b pr / Cy dpTh pr
c43	sc^{28} w^{a}
c44	sc^{29} w^{a}
c45	sc^{45} $\underline{l}$ / sc^{S1} InS
c46	sc^{C} / y sc^{S1} B InS
c47	sc^{Ci}.III / y ac sc pn w spl rb cx ($\underline{sc}^{19i}$)
c48	sc^{H}, TX4 & y f:=
c49	$\underline{l}^{J1}$ sc^{J1} / Del (sc^{7}) 2 & y f:=
c50	$\underline{l}$ sc^{J1} / Del(X)24
c51	$\underline{l}$ sc^{J1} / Del(Xc)Ag (Pontecorvo)
c52	w^{m5L}; sc^{J4}R ♂ & y f:= (w^{m5L}/) ♀
c53	sc^{J6} B & y f:=
c54	sc^{L3}, TX4 (spoon-like)
c55	sc^{L6}
c56	sc^{Mc}, TX3 / y Hw In49 m g
c57	sc^{S1} B In49 ct-$\underline{l}$ lz^{s} / w sn^{5s}
c58	sc^{S1} B In49 lz^{s} / w sn^{5s} bb
c59	sc^{S1} car f In49 v / "plex"
c60	sc^{S1} f In49 v / y ac sc pn w rb cm ct^{6} sn^{3} ras^{4} v m g f car
c61	sc^{S1} f In49 v w & y f:=
c62	sc^{S1} In49 v & y f:=
c63	y sc^{S1} B f In49 v & y f:=
c64	y sc^{S1} B In49 m / oc ptg
c65	y sc^{S1} B In49 sn^{x2} & y f:=
c66	y sc^{S1} B In49 v & y f:=
c67	y sc^{S1} f In49 m & y f:=
c68	y sc^{S1} car f In49 m / oc ptg
c69	sc^{S2}, T12 / Cy
c70	sc^{V1},Inp v / y^{S1} sc^{8} B f In49 v
c71	sc^{V2}, Inh

d. Combinations of scute or similar inversions

No.	Genotype
d1	y sc^{4} B InS w^{a} sc^{8} & y.=
d2	y sc^{4} f w sc^{8} / y Hw In49 m g
d3	y sc^{4} In49 sn^{x2} sc^{8} & y f.=
d4	y sc^{4} In49 w^{a} sc^{8} & y f:=
d5	y sc^{4} In49 v sc^{8} & y f:=
d6	y sc^{4} In49 v w^{a} sc^{8} / w sn^{5} bb
d7	y sc^{4} w sc^{8} (extra Y in ♀)
d8	y sc^{4} B In49 lz^{s} v sc^{S1} / w sn^{5} bb
d9	y sc^{4} In49 v sc^{S1} & y f:=
d10	y sc^{4} InS sc^{S1} (extra Y in ♀)
d11	y sc^{4} InS sc^{S1} / C$\underline{1}$ B
d12	(sc^{6}-sc$^{8-4}$ rein.) $\overline{\text{sc}}^{8}$.Y / sc^{6} B, r't end of X from reinversion 8-4 & sc^{8}.Y / y f:=
d13	sc^{8} B InS w^{a} sc^{4} & y f:=; (sc^{19i}/)
d14	y rein.sc$^{8-4}$ w^{a} InS B & y f:=; sc^{19i} lt cn / Cy lt cn^{2}
d15	sc^{8}.Y / y rein.sc$^{8-4}$ w^{a} InS bb; sc^{19i} / ♂ & sc^{8}.Y / y f:=;(sc^{19i}/) ♀
d16	y rein.sc$^{8-4}$ w^{a} InS f car.Dp(sc^{V1}); sc^{19i} / ♂ & y f:= (sc^{19i}/)
d17	y^{S1} sc^{8} B InS sc^{S1} / w sn^{5s}
d18	y^{S1} sc^{8} B InS y^{3P}
d19	y^{S1} sc^{8} f InS v y^{3P}
d20	y^{S1} sc^{8} InS y^{3P}; Cy / Scd
d21	sc^{-17aH3} f car.sc^{V1}; Cy / sc^{19i}♂ & y f:= Cy / sc^{19i}♀
d22	sc^{L8} sc^{8} & y f:=
d23	sc^{L8} g^{s} v lz^{g} sc^{8} & y f:=
d24	sc^{S1} In49 sn^{x2} sc^{8} & y f.=
d25	sc^{S1} At B In49 sc^{8}
d26	sc^{S1} At In49 v w^{a} sc^{8} & y f:=
d27	("Binsc") sc^{S1} B In49 sc^{8} & y f:=
d28	sc^{S1} B g In49 m sc^{8} & y f:=
d29	sc^{S1} B In49 lz^{s} sc^{8} / y ac sc pn w v g f
d30	sc^{S1} B InS w^{a} sc^{8}
d31	sc^{S1} B InS w^{a} sc^{8} & y f:=
d32	sc^{S1} car B In49 v sc^{8} & y f:=
d33	sc^{S1} car m w^{a} sc^{8} / w In49 lz^{s}
d34	sc^{S1} f In49 v w^{a} sc^{8} & y f:=
d35	sc^{S1} In49 v sc^{8} & sc v f.=
d36	("Binscy") y sc^{S1} B In49 sc^{8} & y f:=
d37	y sc^{S1} B In49 lz^{s} sc^{8} / y ac sc pn w v g f
d38	y sc^{S1} B In49 m sc^{8} & y f:=
d39	y sc^{S1} B In49 sn^{x2} sc^{8} & y f:=
d40	y sc^{S1} In49 sn^{x2} sc^{8} & y f.=
d41	y sc^{S1} B In49 sn^{x2} v sc^{8} & y f:=
d42	y sc^{S1} B In49 v sc^{8} & y f:=
d43	y sc^{S1} B In49 v w^{a} sc^{8} & y f:=
d44	y sc^{S1} f In49 m w sc^{8} / w sn^{5}
d45	y sc^{S1} f In49 v sc^{8} & y f:=
d46	y sc^{S1} f In49 v w^{a} sc^{8} & y f:=
d47	y sc^{S1} g In49 m sc^{8} & y f:=
d48	y sc^{S1} In49 sc^{8}
d49	y sc^{S1} In49 ct$^{\underline{1}}$ v sc^{8} / y v car bb^{-}
d50	y sc^{S1} In49 v sc^{8}
d51	sc^{S1} f In-S y^{3P} & y f:=
d52	sc^{8}.Y / sc^{V1}-v; sc^{19i} / Cy ♂ & sc^{8}.Y / y f:= sc^{19i} / Cy ♀
d53	sc^{V2} B y^{3P}

No.	Genotype
d54	y^{3P} InS sc^{S1} & y f:=; Cy / sc^{19i}
d55	y^{3P} InS sc^{S1} / Del (sc^{7}) 1 & y f:=

e. Translocations of X

No.	Genotype
e1	TX2A124 & y v f car.=
e2	TX3δs1 sc^{S1} B InS w^{a} sc^{8} bb / w sn^{5} bb
e3	TX3δs2 sc^{S1} B InS w^{a} sc^{8} / w sn^{5} bb

TX4 viable & fertile (e4-e19)

No.	Genotype
e4	TX(1B3+)4 sc^{8} B w^{a}
e5	TX(3C2)4 w^{m5} & y f:=
e6	TX(3C2)4 w^{m5} v f bb / w^{m5} C$\underline{l}$ B
e7	TX(5C4)4 y w$^{258-18}$ / y Hw In49 m g
e8	TX(4c3)4 & y f:=
e9	TX(9A1)4 & y f:=
e10	TX(9B&20)4 "W13" / C$\underline{l}$ B
e11	TX(9B&20)4 "W13" car
e12	TX(9B&20)4 "W13" sc v^{m} g / C$\underline{l}$ B
e13	TX(9B&20)4 "W13" y w & y f:=
e14	TX(11A7)4 & y f:=
e15	TX(11B16)4 & y f:=
e16	TX(13B8-9)4 "Sidky a" & y f:=
e17	TX(16A1)4 B^{S} & y v f.=
e18	TX(16A1)4 B^{S}.Y^{S} / sc.$\underline{Y^{1}}$ & y w f.= / sc.$\underline{Y^{1}}$
e19	TX(rt of car)4 & y f:=

f. Altered Y's, sometimes with mutants in X and/or 2

No.	Genotype
f1	Y^{+} / sc w ct f.Y^{S} & y f:=
f2	Y^{+} / w^{a} ct^{6} f.$\underline{Y^{1}}$ (sc^{S1} B InS)
f3	Y^{+} / X^{+}.$\underline{Y^{1}}$ & y w^{a}.=
f4	Y^{+} / y In49 v.$\underline{Y^{1}}$
f5	Y bb^{-} / v; bw^{VA} / Bl L^{2}
f6	Y bb^{-} / y v; bw^{VA} / Bl L^{2}
f7	Y:bw^{+}; bw^{+} (vg, pr) (Y with region of II containing bw inserted in $\underline{Y^{1}}$)
f8	Y:bw^{+} / w^{mR7AH1}; cn bw
f9	Y:bw^{+} / y sc^{4} B InS y^{3P} / X ; cn bw
f10	Y:bw^{+} / y v; bw
f11	Y:bw^{+} / Xc^{2} t^{2}; cn bw
f12	sc^{8}.Y / ac^{3} w^{a}
f13	sc^{8}.Y / $\underline{1}^{J1}$ sc^{J1} & y f:=
f14	sc^{8}.Y / $\overline{\underline{1}}^{J1}$ sc^{J1} pn w rb / sc t^{2} v C$\underline{l}$ B
f15	sc^{8}.Y / sc w B.Y^{S}; Cy / S Sp ab^{2} ltd & y f:=
f16	sc^{8}.Y / sc w ct f.Y^{S} & y f:=; Cy / S Sp ab^{2} ltd
f17	sc^{8}.Y / Xc^{2}
f18	sc^{8}.Y / Xc^{2} t^{2}; cn bw
f19	sc^{8}.Y / Xc^{2} t^{2} & y f:=; cn bw
f20	sc^{8}.Y / Xc^{2} y v & y f:=
f21	sc^{8}.Y / y ac sc B.Dp(sc^{S1}) & y f:=
f22	sc^{8}.Y / y ac sc t^{2} B.Dp(sc^{S1}) & y f:=
f23	sc^{8}.Y / y rein.sc$^{8-4}$ B.Dp(sc^{S1}) & y f:=
f24	sc^{8}.Y / y v
f25	sc^{8}.Y / y v; bw
f26	sc^{8}.Y / y v f.$\underline{Y^{1}}$ & y f:=
f27	sc^{8}.Y / y w.Y^{S} & y f:=

No.	Genotype
f28	sc^{8}.Y♂ & Y:bw^{+}♀ / w^{m4}; cn bw
f29	sc^{8}.Y♂ & Y:bw^{+}♀ / y sc^{8} B In49; cn bw
f30	sc^{8}.Y♀ & Y:bw^{+}♂ / y sc^{8} B In49; cn bw
f31	sc^{8}.Y♂ & Y:bw^{+}♀ / y v; bw
f32	sc^{8}.Y♀ & Y:bw^{+}♂ / y v; bw
f33	sc^{8}.Y♂ & Y^{+}♀ / y v; bw^{VA} / L^{2} $\underline{l}$
f34	$\underline{Y^{1}}$ / f.Y^{S} & sc v f.=
f35	$\underline{Y^{1}}$ / f.Y^{S} & y^{2} wy^{2} g^{2} f.=
Sterilizer ("sz") stocks f36-f42	
f36	("sz^{+}") $\underline{Y^{1c}}$ / X.Y^{S}
f37	("sz bw") $\underline{Y^{1c}}$ / X.Y^{S}; bw
f38	("sz c") $\underline{Y^{1c}}$ / X.Y^{S} & y v f.=; c
f39	("sz e") $\underline{Y^{1c}}$ / X.Y^{S} & y v f.=; e
f40	("sz w") $\underline{Y^{1c}}$ / w.Y^{S}
f41	("sz y e") $\underline{Y^{1c}}$ / y In49 v f.Y^{S}; e
f42	("sz y w") $\underline{Y^{1c}}$ / y w.Y^{S} & y ct^{6} f.=
f43	("facl") $\underline{Y^{1c}}$ / y^{2} oc ptg fu.Y^{S} ♂ & $\underline{Y^{1c}}$ / y^{2} oc ptg B^{M1} / sc^{S1} fu In49 sc^{8} ♀
f44	("Jynd") $\underline{Y^{1c}}$ / w^{e} oc lz^{3}.Y^{S} ♂ & $\underline{Y^{1c}}$ / sc^{J1} pn w rb cm ct^{6} sn^{3} oc ras^{2} v dy g^{2} f od car sw / y sc^{S1} B In49 lz^{s} ♀
f45	$\underline{Y^{1c}}$ / sc w B.Y^{S} & y ct^{6} f.=
f46	$\underline{Y^{1c}}$ / sn^{5} oc ptg.Y^{S} & $\underline{Y^{1c}}$ / y v f.=
f47	$\underline{Y^{1c}}$ / y ct^{6} f.Y^{S} & y w^{a}.=
f48	$\underline{Y^{1c}}$ / y w.Y^{S} & y v f.=
f49	$\underline{Y^{1c}}$ / y^{2} oc ptg fu.Y^{S} & $\underline{Y^{1c}}$ / y w^{a}.=
f50	$\underline{Y^{1c}}$ / y^{2} sn^{5} oc ptg.Y^{S} & $\underline{Y^{1c}}$ / y v f.=
f51	sc.$\underline{Y^{1}}$ / y.Y^{S} & ac w^{a} ct^{6} f.=
f52	sc.$\underline{Y^{1}}$ / ct^{6} mw f.Y^{S} & y f:=
f53	sc.$\underline{Y^{1}}$ / sc w BB^{L},In.Y^{S} & y f:=
f54	sc.$\underline{Y^{1}}$ / sc w B.Y^{S} & y f:=; Cy / S Sp ab^{2} ltd
f55	sc.$\underline{Y^{1}}$ / sc w ct^{6} B.Y^{S} & y f:=
f56	sc.$\underline{Y^{1}}$ / sc w ct^{6} f.Y^{S} & y f:=; Cy / S Sp ab^{2} ltd
f57	sc.$\underline{Y^{1}}$ / w^{a} ct^{6} f.Y^{S} & y f:=
f58	sc.$\underline{Y^{1}}$ / y ac sc ct^{6} f.Y^{S}
f59	sc.$\underline{Y^{1}}$ / y ac sc pn ct^{6} f.Y^{S} & y f:=
f60	sc.$\underline{Y^{1}}$ / y ac sc w^{a} ct^{6} f.Y^{S} & y f:=
f61	sc.$\underline{Y^{1}}$ / y ct^{6} f.Y^{S} / sc^{8} B InS w^{a}
f62	sc.$\underline{Y^{1}}$ / y In49 v f.Y^{S}
f63	sc.$\underline{Y^{1}}$ / y In49 v f.Y^{S}; e
f64	sc.$\underline{Y^{1}}$ / y rein.sc$^{8-4}$.Y^{S} & y f:=
f65	sc.$\underline{Y^{1}}$ / y v ♀ & Y^{+} / y v ♂; bw^{VA} / L^{2} $\underline{l}$
f66	sc.$\underline{Y^{1}}$ / y w.Y^{S} & y f:=
f67	sc.$\underline{Y^{1}}$ / y w$^{m258-18}$ t^{2} v f ♀ & Y^{+} / y w$^{m258-18}$ t^{2} v f ♂
f68	sc.$\underline{Y^{1}}$ / y.Y^{S} & y f:=
f69	sc.$\underline{Y^{1}}$ / y.Y^{S} & y f:=; cn bw; (e)
f70	sc.$\underline{Y^{1}}$ / y^{2} v f.Y^{S} & y w f.=
f71	sc.$\underline{Y^{1}}$ / y^{2} w^{a} ct^{6} f.Y^{S}
f72	sc.$\underline{Y^{1}}$ / y^{2} w^{a} InS f.Y^{S} / y^{2} w^{a} ct^{6} f.Y^{S}
f73	sc.$\underline{Y^{1}}$ / y^{2} X^{+}.sc^{8} w^{a} InS B ♀ & sc.$\underline{Y^{1}}$ / y^{2} w^{a} ct f.Y^{S} ♂ (tandem X.X giving rings)
f74	y^{3}.$\underline{Y^{1}}$ / oc f.Y^{S} & y^{3}.$\underline{Y^{1}}$ / y f:=
f75	("plond") y^{3}.$\underline{Y^{1}}$ / y ac sc pn w rb cm ct^{6} sn^{3} oc ras^{2} v m g^{2} f car / sc^{S1} B In49 lz^{s} ♀ & y^{3}.$\underline{Y^{1}}$ / y^{2} oc lz.Y^{S} ♂
f76	y^{3}.$\underline{Y^{1}}$ / sc w oc f.Y^{S} & y f:=
f77	y^{3}.$\underline{Y^{1}}$ / sc w sn^{5} f.Y^{S} & y f:=
f78	y^{3}.$\underline{Y^{1}}$ / sc^{V1-} oc lz^{3}.Y^{S} & y f:=

No.	Genotype
f79	y^3.$Y^{\underline{l}}$ / sc^{V1-} w.Y^s & y f:=
f80	y^3.$Y^{\underline{l}}$ / w^e oc lz^3.Y^s & y f:=
f81	y^3.$Y^{\underline{l}}$ / y.Y^s & y f:=
f82	y^3.$Y^{\underline{l}}$ / y oc lz.Y^s & y f:=
f83	y^3.$Y^{\underline{l}}$ / y w oc lz^3.Y^s & y f:=
f84	y^3.$Y^{\underline{l}}$ / y^2 w.Y^s & y f:=
f85	y^3.$Y^{\underline{l}}$ / y^2 w^a sn^5 f.Y^s & y f:=
f86	Y^s / g^2 B.$Y^{\underline{l}}$ & y f:= (Stern)
f87	Y^s / y ct^6 f.$Y^{\underline{l}}$ & y f:=
f88	Y^s / y v f.$Y^{\underline{l}}$ & f.=
f89	Y^S.Y^S#2 / y v f.$Y^{\underline{l}}$ & y f:=
f90	sc^{V1}.Y^s / y v f bb.$Y^{\underline{l}}$ & y f:=
f91	sc^{V1}.Y^s / y In49 v B.$Y^{\underline{l}}$ & y f:=
f92	sc^{V1}.Y^s / y In49 v f.$Y^{\underline{l}}$ & y f:=
f93	sc^{V1}.Y^s / y In49 v.$Y^{\underline{l}}$ & y f:=
f94	TY2 (B.N.) / ab^2 bw sp♂ & ab^2 bw sp♀
f95	TY3 $\underline{l}$ (II4Aa2) / tu h D CXF ca
f96	TY4 & y f:= (Edmondson)

g. Chromosome 2

g1	ab^2 / S^2 Ins(CyL,CyR)
g2	ab^2 bw sp / S^2 Cy cn^2 L^4 sp^2 (iso 2, Apr. '49)
g3	ab^2 bw^{5-} mr / S^2 Cy cn^2 L^4 sp^2
g4	ab^2 bw^{VA} / Cy Bl cn^2 L^4 sp^2
g5	ab^2 cn bw^{5-} / Cy cn^2 L^4 sp^2
g6	ab^2 cn InNSR mr / Cy Bl cn^2 L^4 sp^2
g7	ab^2 cn^4 Pm^1 / Cy Bl cn^2 L^4 sp^2
g8	ab^2 InNSR mr / Cy Bl cn^2 L^4 sp^2
g9	ab^2 mr sp / Cy Bl cn^2 L^4 sp^2
g10	al b c sp
g11	al b cn sp
g12	("albasp") al b pr cn vg a sp / Cy cn^2 L^4 sp^2
g13	al^2 Cy Bl cn^2 L^4 bw sp / InsNS px sp
g14	al^2 Cy Bl lt^3 cn^2 L^4 sp^2 / InsNS mr
g15	al^2 Cy,InL lt^3 / b pr Bl lt^3 InCyR L^4 sp^2
g16	ap^4 / Rvd,In^{2LR}
g17	ap^4 cn / S^2 Cy L^4 sp^2
g18	("apl") al dp b pr c px sp / Cy
g19	b InsNSI&R mr / ab^2 InCyR L^4 sp^2
g20	b pr
g21	b pr c px sp
g22	b pr InCyR
g23	Bl bw^{VA} / Cy,InL L^2
g24	Bl L^2 / Cy
g25	Bl stw^3 / IndpT23 b
g26	Bld InCyR / sc^2 pn
g27	bur cn
g28	bw (iso 2,3)
g29	bw^{5-} / Cy cn^2 L^4 sp^2
g30	bw^D
g31	c (iso 2,3)
g32	c bw
g33	cg c / U
g34	cn (iso 2)
g35	cn bw

No.	Genotype
g36	cn bw sp
g37	cn ms2.1 rm / Cy cn^2 L^4 sp^2
g38	cn ms2.1 rm sp / al^2 Cy pr Bl cn^2 L^4 sp^2
g39	cn^2 InCyR cg sp^2 / b IndpT23
g40	cn^2 InCyR cg sp^2 / dp b L^4 Pm^1
g41	cn^2 InCyR cg sp^2 / InsNS px sp
g42	cn^3 cg bw^{5-} mr / Cy cn^2 L^4 sp^2
g43	*crs / al^2 Cy Bl cn^2 L^4 sp^2 (iso 2)
	*crs, located between sp and M(3)33a, denotes the ♂-sterile gene found in the chromosome supposedly containing cr-u. This chromosome had no ♂-sterile at the locus assigned to cr-u itself. In previous stock lists we denoted the present ♂-sterile as cr-u, not knowing of the locus difference.
g44	Cy dp^2 / fr
g45	dp cn bw
g46	dp b cn c a mr / Cy
g47	dp b cn c sp / al^2 Cy Bl cn^2 L^4 sp^2
g48	dp b L^4 Pm^1 / dpTh Cy pr
g49	dp b L^4 Pm^1 / IndpT23 b
g50	dp bw^{5-} mr / S^2 dpTh Cy cn^2 L^4 sp
g51	dp^{02}
g52	dp^{03} ta sp / Cy Bl cn^2 L^4 sp^2 (iso 2)
g53	dp^T / al^2 Cy cn^2 L^4 sp^2
g54	dp^T ab^2 pr Bl rn NSR mr / al^2 Cy cn^2 L^4 sp^2
g55	dp^T Sp cn InNSR mr / S^2 Cy Bl cn^2 L^4 sp^2
g56	dp^T Sp ta cn ms2.1 crs / S^2 Cy pr Bl cn^2 L^4 sp^2
g57	dpTh Cy Bl cn^2 L^4 sp^2 / InNSL InNSR px sp
g58	dp^{V2}
g59	Dt bw
g60	fes ab^2 pr(?) / al^2 InCyL InCyR stl
g61	fes Alu lt / al^2 Cy lt^3 (L^4) sp^2
g62	fes IndpT23 b sp / al^2 Cy L^4 sp^2
g63	fes pr rn / al^2 Cy cn^2 L^4 sp^2
g64	Gla / pi
g65	Gla / S^2 Cy cn^2 L^4 bw sp
g66	In,NSL In,NSR / al^2 Cy,InL lt^3 L^2
g67	InsCyL&R $\underline{l}$ cn^2 / dp b L^4 Pm
g68	j-1 ab^2 NSR mr / Cy cn^2 L^4 sp^2
g69	M$\underline{l}^2$ / Cy cn^2 L^4 sp^2
g70	Mz / In2L
g71	mi sp / Pm^2
g72	rn / Cy cn^2 sp^2
g73	mn / Cy cn^2 L^4 sp^2
g74	mn Pin / Cy cn^2 L^4 sp^2; st ca CG
g75	pr ta cn ms2.1 crs / Cy Bl cn^2 L^4 sp^2
g76	px bw mr sp / ds^{33K} Pm
g77	Px^{2-} / Cy cn^2 L^4 sp^2
g78	rk cn bw (iso 2)
g79	rn / Cy Bl cn^2 L^4 sp^2
g80	rn In2RM / Cy cn^2 sp^2
g81	S dp Sp ab^2 ta cn c mr / dpTh Cy Bl cn^2 L^4 sp^2
g82	S dp^T / al^2 Cy cn L^4 sp^2
g83	S Sp ab^2 bw^{5-} / al^2 Cy cn^2 L^4 sp^2
g84	S Sp ab^2 ap^4 NSR px sp / al^2 Cy Bl cn^2 L^4 sp^2
g85	S Sp ab^2 pr Bl rn T23 / al^2 Cy cn^2 L^4 sp^2
g86	S Sp ab^2 ta cn c mr / dpTh Cy pr Bl cn^2 L^4 sp^2

No.	Genotype
g87	S Sp Bl bw^{D} / Cy cn^{2},InsCy
g88	S Sp Bl L^{2} / Cy cn^{2} sp
g89	S Sp Bl L^{2} Px / dp b L^{4} Pm^{1}
g90	S Sp Bl Pfd bw^{D} / Cy
g91	S Sp(?) Bl vg^{D} bw^{D} / dp^{Th} Cy pr cn^{2} (InsCy)
g92	S Sp (ls^{+}?) cn bw sp / dp^{Th} Cy,InL cn bw sp
g93	S Sp crs / al^{2} Cy Bl L^{4} sp^{2}
g94	S Sp InNSR mr / dp^{Th} Cy Bl cn^{2} L^{4} sp^{2}
g95	S Sp ta cn ms2.1 crs / dp^{Th} Cy Bl cn^{2} L^{4} sp^{2}
g96	S^{2} cn^{2} Ins,Cy / dp^{T} Sp ls cn bw sp
g97	S^{2} Cy Bl cn^{2} L^{4} bw sp / InNSL InNSR px sp
g98	S^{2} Cy Bl cn^{2} L^{4} sp^{2} / InNSL InNSR px sp
g99	S^{2} Cy cn^{2} InCyR sp^{2} / InNSL InNSR px sp
g100	S^{2} Cy cn^{2} L^{4} sp^{2} / b pr Bl tk
g101	S^{2} Cy cn^{2} L^{4} sp^{2} / InNSL InNSR px sp
g102	S^{2} ls Cy,InsL&R cn^{2} bw sp / dp^{T} Sp ab^{2} cn bw sp
g103	S^{2} (ls^{+}) Cy,InL cn bw sp / dp^{T} Sp (ls^{+}) cn bw sp
g104	S^{2} dp^{Th} Cy Bl cn^{2} L^{4} bw sp / InNSL InNSR px sp
g105	sm px pd / al^{2} Cy cn^{2} L^{4} sp^{2}
g106	Sp bu cn InNSR px sp / Cy Bl cn^{2} L^{4} bw sp
g107	stw^{3}
g108	ta cn bw / al^{2} Cy Bl cn^{2} L^{4} sp^{2} (iso 2)
g109	ta cn bw sp / Cy Bl cn^{2} L^{4} sp^{2} (iso 2)
g110	("twelvepl") al dp b pr cn vg c a px bw mr sp / al^{2} Cy pr lt cn^{2} L^{4} sp^{2}
g111	Uf
g112	vg (iso 2,3)
g113	vg bw
g114	vg^{-D} sp^{2} / Cy cn^{2} L^{4} sp^{2}

h. Chromosome 3 (containing genes of X or 2 also in a few cases)

No.	Genotype
h1	bwl (brown-like in chrom.3)
h2	bwl Sb H / ru h D InsCXF ca
h3	C3G #2 (Gowen)
h4	C3G (Pasadena)
h5	$ca^{572jIIIa3}$ / Mé,Ins ri $Sb^{\underline{1}}$
h6	ca^{708k} s-l / Mé,Ins ri $Sb^{\underline{1}}$
h7	Cor / ru h D InsCXF
h8	cv-c sbd^{2}
h9	D tra / InLP Dfd InRP ca
h10	D^{3} H / InsP
h11	D^{3} Sb / InLP Dfd InRP ca
h12	e P^{i} / ru h D InsCXF e
h13	e^{11}
h14	Gl bx^{D} / InsLVM
h15	glass-like
h16	h th st W $\underline{1}347^{(+)}$ cu sr e^{s} / ru h D CXF ca
h17	in
h18	(Cy) $\underline{1}347^{(+)}$
h19	$\underline{1}347$ / ru h D CXF ca
h20	(Cy / Pm) $\underline{1}347$ Sb In3R(Mo) sr / ru h D InsCXF ca
h21	M3w / InRC e $\underline{1}3e$
h22	M3y Gl / InsLVM
h23	Mé,InL bx^{D} / ru h D InsCXF Sb
h24	Mé,InL InRC e $\underline{1}3e$ / ru h D Sb InsCXF

No.	Genotype
h25	Mé, InL Sb / ru h D InsCXF
h26	Mé,InL Sb InRC e 13e / ri bod e^{s}
h27	Mé,Ins ri Sb^{1} / ru h D InsCXF ca
h28	R Ly / InLP gm
h29	ri p^{p}
h30	ri p^{p} Ina (/ru h D InsCXF ca)
h31	ri p^{p} Inc 1 / ru h D InsCXF ca)
h32	ri p^{p} Ind 1 / ru h D InsCXF ca
h33	ri p^{p} sep,Inp
h34	ri sbd e^{2}
h35	("rucuca") ru h th st cu sr e^{s} ca
h36	ru D^{3} st bx^{D} e^{s}(?) / Mé,Ins ri Sb^{1}
h37	ru h D^{3} st ri In3C e 13e / Mé,Ins ri Sb^{1}
h38	ru h ri p^{p} Inb (/ ru h D InsCXF ca)
h39	ru h ri p^{p} Ine(3R) / ri p^{p} sep,Inp
h40	("rupes") ru h th st p^{p} cu sr e^{s}
h41	("ruPrica") ru h th st cu sr e^{s} Pr ca / Mé,T23
h42	ru st C$_{3G}$ e^{s} (iso 3)
h43	ru st C$_{3G}$ sr e^{s}
h44	Sb bx^{D} / Xa,T23
h45	sbd e^{11}
h46	se h
h47	se rt^{2} th / Mé,InL
h48	se ss
h49	ru 3L.DpXhet; Dpy^{+}.3R e^{s} ca / ru h D InsCXF ca separated arms of 3 (Dubinin 1)
h50	(ru h?) T3L.4L;4R.3R(e^{s}?) / 1 InPL Dfd InPR 1 separated arms of 3 (Dubinin 2)
h51	ss
h52	ss e
h53	st
h54	st C$_{3G}$ (iso 2,3)
h55	(b sp/); st C$_{3G}$ sr e^{s} (iso 3 $1 Apr.'49)
h56	st C$_{3G}$ sr e^{s} (iso 3 $2 Apr.'49)
h57	st Sb^{r} e^{s} ro ca
h58	(sp^{2};) suHw^{2} bx bxd / Mé,Ins ri Sb^{1}
h59	th st W 1347 cu sr e^{s} / ru h D InsCXF ca
h60	th st W p^{p}
h61	("threepl") ru h st p^{p} ss e^{s}
h62	tra / Mé,T23
h63	tra p / ru h D CXF e
h64	ve h th
h65	ve R D^{3} bx^{D} (e^{s}?) Pr ca / InPL Dfd InPR ca
h66	ve R D^{3} bx^{D} (e^{s}?) Pr ca / Mé,Ins ri Sb^{1}
h67	ve R D^{3} Sb^{Spi} Bd^{G} / InsP
h68	W Sb / D InsCXF
h69	Xa,T23 ca / e^{s} cd ro cmp ca

i. Chromosome 4

i1	bt^{D} / ci^{D}
i2	Cat / ci^{D}
i3	Cat / gvl ey^{R}
i4	ci ey^{R}
i5	ey^{D} / ci^{D}
i6	four-sim / ci^{D}

No.	Genotype
i7	gvl ey^R
i8	M(4)/sv/sv
i9	TY4 (Edmondson) / four-sim♂ & four-sim♀

j. Multiple Chromosomes

No.	Genotype
X2 (j1-j14)	
j1	("maple-12") y ac sc pn w rb cm ras^2 v g car ♂ & y f:=♀; al dp sc^{19i} b pr cn vg c a px bw mr sp / Cy cn^2 L^4 sp^2
j2	sc w $B^{S2}.Y^S$ & y f:= (B^{S2} Del.-Inser. in 2)
j3	sc^8 B; bw
j4	sc^8 f In49 v; bw^{VA} / L^2 $\underline{1}$ (iso Y,X,2)
j5	("tra") w^e ♂ & y v.= ♀; tra / D InCx
j6	w^e; crs / al^2 Cy cn^2 L^4 sp^2
j7	y f:=; bw^{VA} / L^2 $\underline{1}$
j8	y f:=; cn bw
j9	y f:=; Cy / Gla
j10	y v; bw
j11	y v; bw^{VA} / L^2 1
j12	y v f^x: f^{+i}; bw^{VA} / L^2 $\underline{1}$
j13	y^2 t^2; cn bw
j14	y^{Si} sc^8 B f In49 v; bw^{VA} / L^2 $\underline{1}$
j15	(X3) sn^3; Mw / $\underline{1}$ InLP InRP $\underline{1}$
j16	(X3) y^2; st C3G sr e^S
j17	(X4) y.=; ci ey
j18	(Y2) TY2G / b pr (tk)
j19	(Y3) TY3(II4Aa^3) $\underline{1}$ / ru h D InsCXF ca (TY3 in♀&♂)
j20	(Y3) TY3(II4Ab^2) $\underline{1}$ / ru h d CXF ca / InLP Dfd InRP ca
j21	(Y4) TY,4 / ci ey♂& ci ey ♀
23 (j22-j43)	
j22	"apl" / Cy sp; ru h D InsCXF ca / Sb InRMo
j23	bw; e
j24	bw ss
j25	c; e
j26	c fs / Cy; st C_{3G} (iso 2,3)
j27	c ms / Pm ; st C_{3G} (iso 2,3)
j28	c ; st C_{3G} (iso 2,3)
j29	Cy Bl cn^2 L^4 sp^2 / cn ms2.1 rm sp; ru h D InsCXF / ve th $\underline{1}$
j30	Cy $\underline{1}$ / $\underline{1}$; C3G st ca
j31	Cy / Pm ; st C_{3G} (iso X,2,3)
j32	Cy / $\underline{1}$; ru h D InsCXF ca / InLP Dfd InRP ca
j33	dp b bw^{5-} mr / Cy cn^2 L^4 sp^2; Dl H e P^i /
j34	dp^V ; vo^3
j35	("iser") S Sp (crs) / Cy InL lt^3; Mé,Ins / Bd^G
j36	("Pale dbe") dp b cn (c?) P^- / Cy cn^2; e P^i / e P^i
j37	("Pale dbH") dp b cn (c?) P^- / Cy cn^2; Dl H e P^i / In3R $\underline{1}$
j38	("Pale Indp") IndpT23 b P^-.Dl H e P^i / dp b Pm^1; Sb In3R
j39	Pm / ap^4; ru h D CXF ca / Sb InMo
j40	rn T23 / Cy sp^2
j41	("sifter") S Sp P^- T23.InsCXF / al^2 Cy cn^2 L^4 sp^2; Dl H e P^i
j42	S Sp (crs) / Cy InL lt^3; D^3 tra / Mé,Ins
j43	vg; e^{11}
23 Stocks for mutation rate in 2 & 3 (j44-j56)	
j44	bw; Mé,InL In3RC e $\underline{13e}$ / ru h D Sb InsCXF
j45	bw sp; ri e
j46	bw sp; ru h D^3 ri In3RC e $\underline{13e}$ / Mé,Ins ri $Sb^{\underline{1}}$

No.	Genotype
j47	cn ms2.1 rm sp / Cy Bl cn^{2} L^{4} sp^{2}; h (iso '51)
j48	cn crs / Cy Bl cn^{2} L^{4} sp^{2}; ri (iso '51)
j49	("CySMé") dpTh Cy,InL sp^{2} / S Sp sp^{2}; ru h D^{3} ri InRC e 13e / Mé, InsLR ri Sb^{1}
j50	dp^{T} Sp,ta cn ms2.1 crs / S Cy pr Bl cn^{2} L^{4} sp^{2}; e
j51	sp^{1}; Mé,InL In3RC e 13e / ru h D Sb InsCXF
j52	S Sp sp^{2} / dpTh Cy,InL sp^{2}; e
j53	S Sp sp^{2} / T^{h} Cy,InL sp^{2}; ri e
j54	ta sp / Cy Bl cn^{2} L^{4} sp^{2}; jv (iso'51)
j55	ta sp / Cy Bl cn^{2} L^{4} sp^{2}; ru
j56	ta sp / Cy Bl cn^{2} L^{4} sp^{2}; th (iso '51)
j57	(24) "apl" 1 / Cy cn^{2} sp^{2}; four-sim / ci ey
j58	(24) bw; (e); ci-M^{4} / ci ey / ci ey
X23	(j59-j66)
j59	("eosin sifter") w^{e}; P- T23.InsCXF / Cy L^{4} sp^{2}; Dl H e P^{i}
j60	sc^{8}.Y / y sc^{S1} f In49 v sc^{8}; Cy / S Sp; st C3G
j61	(Marked Inversions, "MI") y^{Si} sc^{8} InS y^{3P}; al^{2} Cy lt^{3} sp^{2} / dp b Pm^{1}; ru h D InsCXF ca / Sb In3R
j62	("Pale") w^{e}; P- / Cy ; P^{i} / P^{i}
j63	y.=; cn bw; e
j64	y In49 v; bw; e
j65	y sc^{S1} f In49 v sc^{8}; bw; e
j66	y^{Si} sc^{8} B f In49 v; bw; e
j67	(X24, "scar") sc t v f car; Cy / bw; ey
j68	(YX2) Y:bw^{+}(sc^{8}.Y) / y v ♂& sc^{8}.Y / y v ♀; S Sp cn bw / dpTh Cy cn bw
j69	(Y23) Y:bw^{+}; Cy,InL rn; InsCXF / IndpT23 b
j70	(Y23) Y:bw^{+}; Mé,T23 / T^{h} Cy cn^{2} bw sp
j71	(X234) y f:=; bw; e; ci ey^{R}

k. Virus

k1 e;CO_2 - sensitive

AMES, IOWA: IOWA STATE COLLEGE

Note: Stock list unchanged. See DIS-24, p. 23.

AUSTIN, TEXAS: UNIVERSITY OF TEXAS

Note: Stock list unchanged. See DIS-22, pp. 24-27.

BALTIMORE, MARYLAND: JOHNS HOPKINS UNIVERSITY

Wild Stocks

11-1 St. Louis - 1
11-2 St. Louis - 2
11-3 St. Louis - 7 (bw)

Chromosome 1

8 lz^{50d}/y

Chromosome 2

17 ap^{49}/Cy
19a M(2)$^{8d.1}$/Cy
20 net S ho/Cy E-S
22 rn/Cy

Chromosome 3

38 p ss bx/T(2,3)Xa

Chromosome 4

43 Ce/ci ey^{R} & ci ey^{R}
44 R(+)15/ci ey^{R}
45 R(ci)24/ci ey^{R}

Multichromosomal

47 Su-er bw; st er
48 b Su-er^{+} bw; st er
52a Ins(1)sc^{S1} B sc^{8} w^{a}/+; Cy/Pm ds^{33k} dp; H/Sb-C

52b Ins(1)sc^{S1} B sc^{8} w^{a}; T(2,3)Xa
53a Cy sp^{2}/Pm ds^{33k} dp; H/Sb-C
53b dp; e
54 pr cn; by

Duplication

10 Dp(sc^{S1})y w f

Inversions

12a car $\underline{l}^{(C1+1)}$/Ins(1)sc^{S1} B sc^{8} w^{a}
12b car $\underline{l}^{(A2+2)}$/Ins(1)sc^{S1} B sc^{8} w^{a}
12c car $\underline{l}^{(B2+6)}$/Ins(1)sc^{S1} B sc^{8} w^{a}
12d car $\underline{l}^{(A3+3)}$/Ins(1)sc^{S1} B sc^{8} w^{a}
12e car $\underline{l}^{(B3+1)}$/Ins(1)sc^{S1} B sc^{8} w^{a}
12g car $\underline{l}^{(B5+2)}$/Ins(1)sc^{S1} B sc^{8} w^{a}
13 Ins($\overline{l}$)sc^{S1} B sc^{8} w^{a}
27 In(2R)bw^{A}/Cy
28 Ins(2L,2R)Cy bw^{V2}/al dp b pr cn c px sp
31 Ins(2LR)bw^{V29}/Cy
32 Ins(2LR)bw^{V30k1}/Cy
33 In(2R)bw^{V30k10}/Cy
34 Ins(2L,2R)Cy bw^{V34}/b vg
35 In(2)b bw^{VDe1}/b lt $\underline{l}$ cn mi sp
36b In(2)bw^{V13}/Cy
42a In(3R)^{G1} & st p^{p} cu sr e^{s}
57 v; In(2R)bw^{V2}/+ & v

Translocations

63 T(2;3)bw^{V5} st & st
64 T(2;3)bw^{V5}st/T(2;3)p^{Gr} st
71 T(2;3)rn/Cy sp
88a T(2;3)^{G5} Sp L^{34}/pr cn; by
88c T(2;3)^{G5} Sp pr cn & pr cn; by
72 T(2;3;4)bw^{V30k18}(Ins 2LR)/Cy

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA

(1) Department of Zoology

Wild Stocks

1 Canton-S (isogenic)
2 Florida-10 (iso '39; contains In(3R)Payne)
3 Kenya
4 Oregon-R (contains Df(2R)c)
5 +3
6 Florida-26 (inbred 24 generations)

Chromosome 1

100 B
101 Bx
102 br w^{e} ec rb t^{4}/In(1)sc^{8} dl-49 y^{31d} w^{a} lz^{s} B
103 car bb (with Y)
104 fa
105 kz g^{2} B/$\underline{y}$
107 Df(1)N^{8}/In dl-49 m^{2} g^{4}
109 sd mc
113 v car
115 wy
117 w
119 w^{co} sn^{2}
120 w^{e} bb/C$\underline{l}$B (with Y)
121 w^{e} bb^{1}/C$\underline{l}$B (with Y)
122 w^{e} fa
123 w^{h}
124A w^{m4}
124B w^{av}
124C w^{m-4000}
124D w^{m-6000}
124E X^{c2}w^{vc}/y Hw dl-49 w m^{2} g^{4} f^{5}
124F X^{c2}w^{vc}/In (High Cornell line)
125 y
126 y ac/$\underline{y}$
127 y ac sn^{3}/yf:
128 y ac sc^{8} w^{a}
129 y ac w^{co} sn^{2}/yf:=
130 y fa^{n}
131 y sc m f^{5}
132 y w
133 In(1)y In(1)w
134 y w spl sn^{2}/y f:=
135 y w^{a} ec cv ct v f/C$\underline{l}$B
136 y sn^{3}
137 y sn^{3} bb/$\underline{\text{B bb}}$
156G y^{2} cv v f
138 y Hw dl-49 v^{o} m^{2} f
140 X^{c2}/y f:=

Chromosome 2

201 a px sp
203 al b c sp/Cy al^{2} lt^{3} L^{4} sp^{2}
206 b cn c bw
207 b cn c sp
208 b pr c
210 Bl L^{2}/Cy sp^{2}
212 bw
214 cn bw
216 c
218 cl
220 en
222 l(2)gl cn bw/Cy cn bw
226 L^{4}
226A L^{4} (with modifiers)
228 pr en
230 tr/$\underline{l}$(In)

232 vg
233 vg^{ni}
234 vg^{no}
235 vg^{nw}/sm Cy

Chromosome 3

326G Bd^{G}
301 cu
308 Gl Sb/LVM
310 h (iso)
311 h th st cu sr e^{s} ca
312 Ly/D^{3}
313 Pc/T(2;3)Mé
314 Pr/In(3R)C e
327G ro Bd ca/In(3R)C l(3)a
316 ru h st p^{p} ss e^{s}
328G ru h c cd ca
320 se h
321 se ss
323 ss
324 ss^{a}

Chromosome 4

400 ar/ey^{D}
401 bt
402 bt^{D}/ci^{D}
403 ci
404 ci^{W}
405 ci^{W} (iso)
407 ci ey
408 ci ey^{R}
409 ci gvl ey^{R} sv^{na}
410 ci sv^{na}
411 ci^{D}/Cat
412 ey^{2}
413 ey^{D}/sv^{dl}
414 ey^{D}/Scn
415 spa

Multichromosomal

501 ClB; Cy/Pm ds^{33k}; H/In(3R)Mo Sb sr
505 y; bw; e^{4} ro; sv^{n}
506G y; bw; e^{4} ro; ey^{2}
509 v; pr en
510 y ac sn^{3}; en
513 w; vg
517 bw; e
519 vg; se
521 se h; ci ey^{R}
401A bt; y
408A ci ey^{R}; y
415A spa; y

Deficiencies

107 Df(1)N^{8}/In dl-49 m^{2} g^{4}
600G Df(1)y-svr/Dp(1;f)101

Translocations

601 B bb bb^{12}
603 T(1;2)Bld/ClB
605 g^{2} B X.Y' Y".y Y"
606 T(2;3)E/Cy; D
313 T(2;3)Mé/Pc
607 T(2;3)Xa/Sb bx^{D}

Suppressors

650 dxst Su dx
652 Su-dx dx
656 su-t (t)

(2) Division of Genetics

About 10 wild-type stocks maintained by brother-sister mating.

CHAPEL HILL, NORTH CAROLINA: UNIVERSITY OF NORTH CAROLINA

Note: Special stocks only are listed here.

1 f^{5} su-f
3 S^{W} Cy/Pfd L^{2}
4 S Pfd/al^{2} Cy lt^{3} L^{4} sp^{2}
5 S L^{4}/Cy Pfd
6 cu (gamma-rayed via 2 mosaics)
9 M(3)y Gl/Sb bx^{D})
10 M(3)y Sb/Gl bx^{D}) for crossover selector matings (autoclassifying)
14 ru h th st sr e^{s} ca
16 M(3)y Gl/LVM
17 M(3)y Sb/LVM
18 M(3)y bx^{D}/LVM
19 Gl Sb/LVM
20 Gl bx^{D}/LVM

CHICAGO, ILLINOIS: CHICAGO MEDICAL SCHOOL

Various "tumorous" strains, as follows:

1 Tumorous head, from Gardner, Logan, Utah
2 Chicago wild-type, from Univ. of Chicago
3 Lausanne-Special, from Univ. of Minnesota
4 Oregon-R, Univ. of Minnesota
5 Brown-Scarlet Special, Univ. of Chicago
6 $\underline{l}$(1)7/dl-49 y Hw m^2 g^4)
7 tu^S)from Burdette, Louisiana State
8 st sr e^s ro ca; tu 36a)
9 +♂/$\underline{f}$♀
10 aa tu-36^e
11 $\underline{l}$(1)7/y Hw

CHICAGO, ILLINOIS: ILLINOIS INSTITUTE OF TECHNOLOGY

Note: Stocks included here are not found elsewhere

Wild Stock

1 Chicago '51

Chromosome 2

2 ang
3 tu^{50j}

COLUMBIA, MISSOURI: UNIVERSITY OF MISSOURI

Note: Previous stock lists are cancelled. Only stocks not commonly found in other lists are included here.

g. Inversions

g^2 InEN y/y f:=
g^9 Ins sc^8 EN f/yf:=

i. Closed-X

i5 X^{c2} y^{49}/In49 y Hw m^2 g^4
i9 X^{c2} InAB/y f:=
i15 X^{c2} w^{vc}/sc^7 InAM (stable)
i16 X^{c2} w^{vc}/sc^7 InAM (unstable)

m. Y^{S}X·Y^L (X·Y) and derivatives

m1 X·Y (homozygous)
m8 X·Y InX·Y24 B f v/y f:=
m14 X·Y/$\underline{y^2\ su^{wa}\ w^a\ bb}$
m15 X·Y B f v/$\underline{y^2\ su^{wa}\ w^a\ bb}$
m16 X·Y B y/$\underline{y^2\ su^{wa}\ w^a\ bb}$
m25 Ins 24L+A2R y/$\underline{y^2\ su^{wa}\ w^a\ bb}$
m26 Ins 24L+A2R y v/$\underline{y^2\ su^{wa}\ w^a\ bb}$

n. Duplications

n5 y sc^4 f m·B^S/sc^7 InAM

o. Compound X's other than attached-X

o1 sc^4 + EN·Y^{Lsc8} y sc^4 car m w^a + car$^+$ m$^+$ w$^+$ y·y$^+$/X·Y Bf v
o2 Tandem double X as above, but without y$^+$
o3 Normal X + sc^8, y w + f(?) v cv sc^8 (Double X)
o10 Hw f/X·Y B y (Tandem attached X)

p. Triploids

p1 $\underline{y\ v\ f\ car}$/Muller 5
p2 with free X's

COLUMBUS, OHIO: OHIO STATE UNIVERSITY

Wild Stocks

a1 Chicago
a2 Oregon-R

Chromosome 1 (X)

b1 B
b2 bb/Y^{bb}
b3 Bx^3
b4 car
b5 cm
b6 ec ct^6 v g^3/C$\underline{1}$B
b7 f
b8 g^2
b9 g^2 tv/$\underline{y}$
b10 m
b11 Muller-5 (sc^{S1} B In-S w^a sc^8)
b12 pn^2
b13 ras^4 x y f:=
b14 rb^{48a}
b15 sc
b16 sc ec cv ct^6 v g^2 f car bb$^{\underline{l}}$/C$\underline{1}$B
b17 su$^{S2-v-pr}$/$\underline{y}$
b18 v^{48a}
b19 w

b20 w m f
b21 w^{a}
b22 w^{bf2}
b23 w^{e}
b24 y

Chromosome 2

c1 b
c2 bw
c3 cn
c4 dp
c5 L
c6 L^{2}
c7 L^{r}
c8 M(2)173/Cy al^{2} lt^{3} L^{4} sp^{2}
c9 mi sp/Pm^{2}
c10 pu
c11 vg

Chromosome 3

d1 bx^{34e}/Payne Dfd ca
d2 cd
d3 dl^{7}/In(3R)Mo, Sb sr
d4 e^{11}
d5 ru h th st cu sr e^{s} ca (ru cu ca)
d6 se
d7 ss^{a}
d8 st

Chromosome 4

e1 ey^{2}
e2 spa^{Cat}/ci^{D}

Multichromosomal

f1 f; se (1;3)
f2 m; bw (1;2)
f3 L; se (2;3)
f4 v; st (1;2)
f5 vg; cd (2;3)
f6 w; dp (1;2)
f7 y; bw (1;2)
f8 y; e^{11} (1;3)
f9 y; pu (1;2)
f10 Cy/Pm, ds^{33k}; H/ In (3R) Mo, Sb sr (2;3)
f11 Muller-5; Cy/Pm, ds^{33k}; H/ In(3R)Mo, Sb sr (1;2;3)
f12 Muller-5; Cy/Pm, ds^{33k}; H/ In(3R)Mo, Sb sr; spa^{Cat}/ ci^{D} (1;2;3;4)

Translocation

g1 T(1;2)7/C$\underline{l}$B

Inversions

delta-49	In(1)dl-49	Stocks no. h11
C$\underline{l}$B	In(1)Cl	Stocks no. b6, b16
Müller-5	In(1)sc^{S1} and In(1)S	Stocks no. b11, f11, f12
Cy	In(2L)Cy and In(2R)Cy	Stocks no.c8,f10,f11,f12, i27
Pm	In(2LR)Pm	Stocks no. f10,f11, f12,i27
Pm^{2}	In(2LR)Pm^{2}	Stock no. c9
Payne Dfd ca	In(3L)P and In(3R)P	Stocks no. d1, i16-i26
	In(3R)Mo	Stocks no. d3, f10, f11, f12, i27

Special Stocks

h1 sc·$Y^{\underline{l}}$/y w·Y^{s} x y f:=
h2 sc^{8}·Y/y v
h3 $Y^{s}Y^{s}$#2/y v f· $Y^{\underline{l}}$x y f:=
h4 $Y^{s}Y^{s}$#3/y v f· Y x y f:=
h5 sn^{3} lz^{BS} v/ClB
h6 sn^{3} lz^{46} ras^{4} v/C$\underline{l}$B
h7 sn^{3} lz^{g} v/$\underline{y\ w\ f}$
h8 $lz^{BS,46}$ ras^{4} v/C$\underline{l}$B
h9 $lz^{BS,g}$ v f/$\underline{y\ w\ f}$
h10 $lz^{46,g}$ v/$\underline{y\ w\ f}$
h11 sn^{3} $lz^{BS,46,g}$ v f/dl-49, (y Hw) m^{2} g^{4}

Isogenic Stocks

Schultz Oregon-R isogenic series I, X, and R:

i1 Ore-R (I series)
i2 Ore-R ex w (X series)
i3 cv Ore-R(I series)
i4 cv Ore-R (R series)
i5 f Ore-R (I series)
i6 f Ore-R)R series)
i7 f Ore-R (X series)
i8 rb^{S1}
i9 rb^{S2}
i10 v Ore-R (I series)
i11 v Ore-R (X series)
i12 v Ore-R (R series)
i13 y Ore-R (I series)
i14 y Ore-R (R series)
i15 f rev^{+}/f X f (X series)

Special isogenic stocks: Original series, derived in 1947, included type stock and mutants induced on type background by X-ray. Series 1 and 2 derived in 1949 by crossing original rb v with original type and re-extracting. Series 4 derived in 1951 by similar procedure and made isogenic. All Payne DfD ca/$\underline{l}$(3)F for chromosome 3.

i16 Type 1	i18 Type 4	i20 rb 4	i22 v 2
i17 Type 2	i19 rb 1	i21 v 1	i23 v 4

124 rb v 1
125 rb v 4
126 y Payne Dfd ca/l(3)F x Type ♂ in each generation
127 rb v 4; Cy/Pm, ds^{33k}; M/In(3R)Mo, Sb sr

EVANSTON, ILLINOIS: NORTHWESTERN UNIVERSITY

Wild Stocks

Oregon-R
Evanston, Ill. (1949)
La Crosse, Wis. (1949)
Wilmette, Ill. (1950)

Mutant Stocks

Over 125 mutant stocks obtained from various laboratories and still maintained by them.
40 sex-linked lethals, 23 of which have been localized.

KINGSTON, RHODE ISLAND: UNIVERSITY OF RHODE ISLAND

Note: Only one stock of more than routine note: Chromosome 2, bw mt^{A} vg. A high-tumor-incidence stock derived from bw tu.

KNOXVILLE, TENNESSEE: UNIVERSITY OF TENNESSEE

Wild Stocks

Big Ridge, Tennessee (1948)-single female strain
Bikini Atoll (1947)
Gatlinburg, Tennessee (1947)
Great Smoky Mountains, Tennessee (1950-51)-single female strains taken at various altitudes
Oregon-R

LOS ANGELES, CALIFORNIA: UNIVERSITY OF CALIFORNIA, DEPARTMENT OF ZOOLOGY

Wild Stocks

1 Canton-S
2 Oregon-R
3 Oregon-R-C (Df(2)Ore)

Chromosome 1

4 B
5 lz/ClB
6 v
7 y
8 y^{2} cv v f
9 w
10 w m f
11 w^{a}
12 w^{e}
13 +/sc^{2} pn

Chromosome 2

14 b
15 b pr c
16 b vg
17 Bl L^{2}/Cy bw sp^{2}
18 bw
19 bw^{2b}
20 bw^{4}
21 bw^{47j}/Cy bw sp^{2}
22 bw^{D}
23 bw^{DE}
24 bw^{DN}/Cy L^{2} sp^{2}
25 c
26 c bw
27 c bw^{D}
28 c bw^{D6}/In(2LR)40d
29 c bw^{D8}/In(2LR)40d
30 c bw^{D9}/In(2LR)40d
31 cl
32 dp
33 dp bw^{a}
34 lt std/Cy sp^{2}
35 pk bw
36 pr
37 px
38 px bw sp^{2}
39 stw^{48} blt tuf (sp?)
40 vg^{nw} Hia/T(2;3)S^{M} Cy
41 IICZ
42 IIDC
43 IIDD
44 IIDE
45 IIDF

Chromosome 3

46 Bar^{3}
47 cu
48 e^{s}
49 e^{4} wo ro
50 Ly Sb/LVM
51 in pP
52 se
53 st
54 st^{sp}
55 th st cp
56 tx

Multichromosomal

57 B bb; stw
58 b; se
59 b; tx

60 bw; by
61 bw; st
62 bw; st^{sp}
63 bw^{2b}; st^{sp}
64 bw^{a}; st^{sp}
65 Cy sp^{2}/Pm, dp b; DcxF, ru h ca/
In(3R)Mo, Sb sr
66 v; bw

Deficiencies

67 Df(2)bw^{5} sp/Xa
68 cn Df(2)vg^{S}/Cy al^{2} lt^{4} L^{4} sp^{2}
69 Df(2)vg^{B}/Cy L^{4} sp^{2}

Inversions

70 In(1)sc$^{260-22}$
71 In(1)sc$^{260-22}$, In(1)dl-49 v,
In(1)B^{M1}
72 y^{31d} In(1)sc^{8}, In(1)dl-49
73 In(2)bw^{VI}, l/Cy bw sp^{2}
74 b In(2)bw^{VDe1}/b lt l cn mi sp
75 In(2LR)IIA/Cy sp^{2}
76 In(2LR)IIBI/Cy sp^{2}
77 In(2LR)IICA/Cy sp^{2}
78 In(2LR)IICQ/Cy sp^{2}
79 In(2LR)IICR/Cy sp^{2}
80 In(2LR)IICX/Cy sp^{2}
81 In(2LR)IIDH/Cy sp^{2}
82 In(2LR)IIP/Cy Sp^{2}
83 In(2LR)IIV/Cy sp^{2}
84 In(2LR)40d/Cy sp^{2}

Translocations

85 T(Y;2)bw^{D4} c bw^{D4}/In(2LR)40d
86 T(Y;2)IIAU/Cy sp^{2}
87 T(2;3)bw^{D11} c bw^{D11}/In(2LR)40d
88 T(2;3)IIAH/Cy sp^{2}
89 T(2;3)IIAV/Cy sp^{2}
90 T(2;3)IICK/Cy sp^{2}
91 T(2;3)IICS/Cy sp^{2}
92 T(2;3)IIDA/Cy sp^{2}
93 T(2;3)IIDG/Cy sp^{2}
94 T(2;3)IIDJ/Cy sp^{2}
95 T(2;3)IIG/Cy sp^{2}

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY, OSBORN ZOOLOGICAL LABORATORY

Wild Stocks

1 Canton-S
2 Canton-S-C (highly inbred)
3 Cockaponsett Forest, Conn.
4 IF-37, Idaho Falls, Idaho
5 New Britain, Conn.
6 New Haven, Conn.
7 Oregon-R
8 Swedish-b

Chromosome 1 (X)

9 B
10 B-reverted
11 bi
12 bi ct^{6} g^{2}
13 Bg^{49h}/M5
14 Co
15 fu/M5 x fu
16 g^{50e}/y f:=
17 Hw^{49c}/M5
18 In(1)rst^{3}/rst^{3}
19 pn^{2}
20 sc^{7} AM, ptg^{4}/y^{2} w^{a} cv v f
21 sc^{10} w^{a}
22 sc^{S1} B InS, w^{a} sc^{8} (M5)
23 sn^{3}
24 sn^{3} v B
25 sn^{37b}/y^{2} w^{a} cv v f
26 v
27 w
28 w ec
29 w fa
30 w m f
31 w spl
32 w^{a}
33 w^{bf} lz/ClB
34 w^{bl}
35 w^{e}
36 y^{2} w^{a} cv v f (extra Y's)
37 y^{2} w^{a} cv v +^{f}/y f:= (reversion of f)
38 y^{2} v f

Chromosome 2

39 al
40 al b c sp^{2}
41 b vg
42 bs^{2}
43 bw
44 bw bs^{cy}
45 cn
46 cn bw
47 dp
48 dp bw^{a}
49 dp^{vo}
50 hy/Cy, L^{4} sp^{2}
51 L^{2}/Cy, sp^{2}
52 lt std/Cy, sp^{2}
53 ltd
54 ltd^{37b}
55 ltd^{37b} vg
56 pr
57 sca
58 vg

Chromosome 3

59 Dl^{K}/Sb
60 e
61 Gl Sb/In(3)LVM
62 ru h th st cu sr e^{s} ca (w^{a}/w^{a})
63 ss
64 ss^{a}

Chromosome 4

65 ey^{2}
66 sv^{n}

Multichromosomal

67 ct^{461} v; bw; e
68 g; cn
69 g; bw
70 v; bw
71 v; bw; e
72 v; bw; e; ey^{461}
73 v; e
74 w; e
75 w^{a} v; e
76 w^{e}; cn
77 y v f; bw
78 bw; e
79 cn bw; e
80 dp; e
81 pr; ss
82 e; ey^{2}

Attached-X

y^{2} w^{a} cv v f (20, 25)

Double-X

y f:= (16, 37)

Deficiencies

Chromosome 1 (X)

83 N^{8}/dl-49, y Hw m^{2} g^{4}
84 N$^{C-7-179}$/M5
85 N^{45e}/dl-49, y Hw m^{2} g^{4}
86 N^{48j}/M5
87 N 264-8 g^{4}/dl-49, y Hw m^{2} g^{4}
88 N 264-34 T(1;3L)/dl-49, y Hw m^{2} g^{4}
89 N 264-40/dl-49, y Hw m^{2} g^{4}
90 1(1)49c-B5/M5
91 Df(1)y sc (Vogt)/M5
92 Df(1)w (Vogt)/M5
93 y w258-11/dl-49, y Hw m^{2} g^{4}
94 y w258-14/dl-49, y Hw m^{2} g^{4}
95 y w258-14/sc^{7} AM, ptg^{4}
96 y w258-21/dl-49, y Hw m^{2} g^{4}
97 y w258-21/sc^{7} AM, ptg^{4}
98 y w258-42/dl-49, y Hw m^{2} g^{4}
99 y w258-42/sc^{7} AM, ptg^{4}
100 y w258-48/dl-49, y Hw m^{2} g^{4}
101 y w258-48/sc^{7} AM, ptg^{4}
102 y Df(1, t-lz)282-1/dl-49,
y Hw m^{2} g^{4}
103 y Df(1, v)267-4 B/dl-49,
y Hw m^{2} g^{4}
104 Df(1)B263-20/In(1)AM

Chromosome 2

105 Df(2)vg^{B}/Cy, L^{4} sp^{2}
106 Df(2)vg^{C}/Rvd
107 Df(2)vg^{D} sp^{2}/Cy, L^{4} sp^{2}
108 Df(2)vg^{S} cn/Cy, al^{2} lt^{3} L^{4} sp^{2}

Duplications

109 Dp(1;f)X^{c2}/y 1(1)7/y 1 (1) 7
B......(9, 22, 33)
Co.....(14)
Hw.....(17, 83, 85, 87, etc.)

Inversions

In(1)AM...............(104)
In(1)ClB..............(33, 110)
In(1)dl-49............(83, 85, 87, etc.)
In(1)rst^{3}............(18)
In(1)sc^{7}, AM.........(95, 97, 99,etc.)
In(1)sc^{10}............(21)
In(1)sc^{S1}, InS, sc^{8}..(13, 17, 22)
In(2L+2R)Cy...........(105,107,108)
In(2LR)Rvd............(106)
In(3L)P, LVM..........(61)

Translocations

110 T(1;2)42i, w^{a} cy/ClB
111 T(1;4)Al, "CRB" (homozygous)
T(1;3L)N264-34...(88)

NEW ORLEANS, LOUISIANA: LOUISIANA STATE UNIVERSITY

Note: Only tumor stocks are listed here.

1 l(1)7/dl-49 y Hw m^2 g^4
2 l(1)7e l(1)7/dl-49 y Hw
3 lz^3 f
4 tu^h
5 w^e sn Bl-t/sc^{S1} (InS) w^a ct sc^8
6 y l(1)7/dl-49 y Hw m^2 g^4
7 bw tu
8 Hx
9 st sr e^s ro ca; tu^{36a}
10 tu^g
11 tu^{48j}
12 aa tu^{36e}
13 se e^{11} tu^{49h}
14 tu^{wps}
15 bw st tu
16 ed Su^2-dx
17 f^{257-19} B/In AM
18 w^{bf} f^5
19 tu^{50d}
20 vg mt^A bw
21 y B^{265-43} (homozygous)
22 vg bw tu

PHILADELPHIA, PENNSYLVANIA: INSTITUTE FOR CANCER RESEARCH and LANKENAU HOSPITAL RESEARCH INSTITUTE

Wild Stocks

a1 Canton-S
a2 Florida-9
a3 Lausanne-S
a4 Oregon-R-S
a5 Seto Japan
a6 Swedish-b-6
a7 Tuscaloosa
a8 Urbana-S

Chromosome 1

b1 Ax
b2 car bb
b3 dim/dl-49, y Hw m^2 g^4
b4 Dp(1;1)Co
b5 Dp(1;1)Co; Df(1)rst^2/y w bb
b6 gt bb^{11}/ClB
b7 gt w^a spl
b8 In(1)y^4, cv v f
b9 sn^2
b10 rb cx
b11 spl rb^{S2}
b12 su-w^a w^a
b13 svr su-w^a w^a
b14 svr w^a
b15 w
b16 w^{a2}
b17 w^{a3}
b18 w^{a4}
b19 w^{bf} f5
b20 w^{bl}
b21 w^{col}
b22 w^e
b23 w^{e2}
b24 w^h
b25 w^{sat}
b26 w^t fw
b27 y sn^3 bb/B bb
b28 y spl rb^{S2}
b29 y w spl
b30 y^2 su-w^a
b31 y^2 su-w^a w
b32 y^2 su-w^a w^a
b33 y^2 su-w^a w^a spl
b34 y^2 su-w^a w^{a2}
b35 y^2 su-w^a w^{a4}
b36 y^2 su-w^a w^{bf}
b37 y^2 su-w^a w^{bl}
b38 y^2 su-w^a w^{co}
b39 y^2 su-w^a w^{col}
b40 y^2 su-w^a w^h
b41 y^2 su-w^a w^{sat}
b42 y^2 su^2-w^a w^a
b43 y^2 w^a
b44 y^2 eq; Df(Y)Y^{-bb}
b45 y w bb; Dp(1;f)135, y^2

Chromosome 2

c1 al b c sp^2
c2 al dp pr (px)
c3 b cn bw
c4 b lt bw
c5 b lt L^4 bw
c6 b pr c
c7 Bl/esc
c8 bw^D
c9 D^3 H/Payne
c10 Df(2)bw^5/Cy dp^2
c11 G^{rev}/Cy dp^2
c12 In(2LR)Gla/Cy
c13 Df(2)MS8/Cy, al^2 lt^3 Dp(2;2)41 L^4 sp^2
c14 Df(2)MS10/Cy pr, Dp(2;2)41
c15 Df(2)rl^{10a} lt cn/Cy
c16 Df(2)rl^{10b} lt cn/Cy, al^2 lt^3 L^4 sp^2
c17 dp pr px
c18 ds S G b pr/al^2 Cy lt^3 L^4 sp
c19 dw-24F/Cy sp^2

c20 l(2)mat/Cy
c21 L^{2}
c22 lt^{3} Dp(2;2)41 L^{4} sp^{2} In(2R)Cy/Pm^{1}, ds^{33k}
c23 Pm^{2}/mi sp^{2}
c24 Pin
c25 stw^{3}
c26 v/cg c

Chromosome 3

d1 D^{3} ri p^{p} H/Payne
d2 dwh/Payne Dfd ca
d3 Gl Sb H/Payne
d4 in p^{p}
d5 ri p^{p}
d6 ru cu ca Pr/T(2;3)Me
d7 ru cu ca ru h th st cu sr e^{s} ca
d8 Ser/In(3R)c, e l(3)e
d9 Tiny/+

Chromosome 4

e1 ar/ey^{D}
e2 bt
e3 Cat/ci^{D}
e4 sv^{n}

Closed-X

f1 X^{c}, y/f B
f2 Dp(1;1)X^{c2}/y l(1)7/y l(1)7

Heterochromatin Rearrangements

Inversions

g1 In(1)sc^{4}, y sc^{4}
g2 In(1)sc^{L8}, sc^{L8} car m w^{a}/y w dl-49 lz^{sp}
g3 In(1)sc^{8}, w^{a}
g4 In(1)y sc^{4}. sc^{8} (bb^{Def} G); Y's extra
g5 In(1)w^{m4} Oregon-R-1
g6 In(1)w^{m4}; Oregon-R autosomes; Y's extra
g7 In(1)w^{m4w}
g8 In(1)w^{m4w}; Y^{su-Y} extra
g9 In(1)w^{m4w}; Df(Y)Y^{-bb}
g10 In(2)41.59, bw^{D}/Cy

Translocations

h1 In(1)sc^{4}.sc^{8}, y; Cy, bw^{V34k}/ds rl; Y's extra
h2 Y^{S}X.Y^{L}, +/y^{2} su-w^{a} w^{a} bb
h3 ab^{2}/T(Y;2)E
h4 y; bw/T(Y;2)E
h5 Pm'/T(Y;2)G
h6 px bw sp/T(Y;2)J
h7 T(1;2)lt/In(1)sc^{8} dl-49, y^{31d} w^{a} lz^{sp} B; Cy
h8 T(1;2)N264-10/y w dim; y's extra
h9 T(1;2)y N264-10/y w dim
h10 T(1;4)w^{m5}/W; ci ey^{R}
h11 T(1;4)w^{m5} L.w^{VD3} R/y^{2} w^{a} Co; ci ey^{R}
h12 T(1;4)N^{8a} L.w^{m5} R/ci ey^{R}
h13 T(1;4)w^{VD3}/Ins(1)dl-49, sc^{8}, y^{31d} w^{a} B; y's extra
h14 T(1;4)w^{VD3}/y^{2} w^{a} Dp(1;1)Co
h15 T(1;4)w^{VD3}/y^{2} w^{a} Dp(1;1)Co; ci ey^{R}
h16 T(1;4)w^{VD3} L.w^{m5}R/y^{2} w^{a} Dp(1;1)Co; ci ey^{R}
h17 T(1;4) y w258-18/y w dim
h18 T(1;4) y w258-18 L.w^{m5} R/ci ey^{R}
h19 T(2;3)bw^{VD3}, ru h st ca/Cy; ru h st ca
h20 bw^{D} T(2;3)289/Cy; In(3R)Mo, Sb sr
h21 T(2;4)ast^{v}/Cy, al^{2} lt^{3} L^{4} sp^{2}
h22 In(1)w^{m4}; T(2;4)su-var 2/Cy cn^{2}
h23 T(2;3;4)295, bw^{D}/Cy; H
h24 T(Y;3)In(1)w^{m4}; Y's extra
h25 T(Y;3)^{40K}; In(1)w^{m4}; rl

Multichromosomal Stocks

i1 B, b pr c
i2 v; bw
i3 w^{a}; D Cx/tra
i4 In(1)w^{m4}, y^{14d}; Cy/+; Y's extra
i5 In(1)w^{m4}; Cy/T(Y;2)G
i6 y; bw; e; ci ey^{R}
i7 Cy/Pm', ds^{33K}; H/In(3R)Mo, Sb sr(2;3)
i8 L^{2}/+; Sb/+

Enhancers and Suppressors of Variegation

j1 In(1)E-Var 1, y w Dp(1;1)Co/y w dim
j2 In(1)w^{m4}; E-var 3/Pm' (Pm' lost)
j3 In(1)w^{m4}; E-var 4/Cy
j4 In(1)w^{m4}; E-var 5/Cy
j5 In(1)w^{m4}; E-var 6
j6 In(1)w^{m4}; E-var 7/Cy
j7 In(1)w^{m4}; E-var 8/Cy
j8 w; Cy/E-var 8^{46}g
j9 w; Cy/E-var 8^{46}g; w^{vco}/Cx
j10 In(1)sc^{8}, sc^{8} w^{a}; Cy L^{4}/E-var 8^{46}g
j11 In(1)w^{m4}; E-var 8^{46}g/Cy
j12 w^{m4}; E-var 8^{46}g/Cy; Y & w^{m4} X YY; Cy/E-var 8^{46}g
j13 In(1)w^{m4}; E-var 8^{46}g/Cy; Y^{bb} su-var 5
j14 In(1)w^{m4}; E-var 11/+
j15 In(1)w^{m4}; E-var 12/D Cx (Cy/+)

j16 In(1)w^{m4}; E-var 12/D Cx (Cy/+); Y's extra
j17 In(1)w^{m4}; E-var 13/D Cx (Cy/+)
j18 In(1)w^{m4}; E-var 15
j19 In(1)w^{m4}; E-var 15; Y's extra
j20 In(1)w^{m4}; E-var 16
j21 In(1)w^{m4}; E-var 17/+
j22 T(1;2;3)E-var 18, y w dim/y w dim/y w dl-49, lz^{sp}
j23 T(1;2;3)E-var 19, y w Dp(1;1)Co/In sc^8, dl-49 y^{31d} w^a lz^{sp} B
j24 y w bb & In(1)w^{m4} Y^{su-var}
j25 In(1)w^{m4}; Cy/+; Y^{bb} su-var 5
j26 In(1)w^{m4}; T(2;4) su-var 2/Cy
j27 bw^D/Cy, al^2 lt^3 Dp(2;2)L^4 sp^2; su-var 4/+
j28 T(Y;3)su-var 3; Cy/T(2;3)bw^{VD3}, ru h st ca
j29 In(1)w^{m4}, Ore R-2 XYY^{red}
j30 bw^D/+; Su-283/LVM

3 N

k1 bb/+ & + (Oregon-R and Canton-S ancestry)

Attached X's heterozygous for inversions

11 $\frac{\text{y w In dl-49 bb}}{\text{y w bb}}$ & y^2 su-w^a w^a

12 $\frac{\text{In } y^4\text{, w v f bb}}{y^2 \text{ su-}w^a\ w^a\ \text{bb}}$ & Dp(1;1)Co

13 yf:= bw, e, ci ey^R

Special stocks isogenic with each other and with Oregon-R

Three strains: I, derived from crosses in 1947 to inbred (brother-sister) stock from Oregon-R-S, inbred since then; X from backcrosses of I strains to inbred Oregon-R (re-extracted,1948); R from extraction from crosses to Oregon-R (I) after recombination of mutants in p19 y cv v f stock. A strains represent mutations within R strains, B strains mutations within I strains.

p1 Oregon-R (I)
p2 Oregon-R ex w (X)
p3 cv Oregon-R (R)
p4 f Oregon-R (R)
p5 f^{rev} +/f♀ x f♂ (X)
p6 f^{+} 51i/f♀ x f♂ (A)
p7 gt Oregon-R (I)
p8 gt w^a Oregon-R (I)
p9 M998/Oregon-R♀ x Oregon-R♂ I (B)
p10 rb^{s1} Oregon-R (B)
p11 rb^{s2} Oregon-R (B)
p12 v Oregon-R (I)
p13 v Lausanne (I)
p14 v Oregon-R (R)
p15 w^{48h} Oregon-R (B)
p16 y Oregon-R (I)
p17 y Oregon-R (R)
p18 y w^{51a} Oregon-R (A)
p19 Ore-R bb Y & Ore-R from p1 each generation (I)
p20 Ore-R bb Y & y cv v f (recombined from I strains)

SALT LAKE CITY, UTAH: UNIVERSITY OF UTAH

Wild Stocks

1 Canton-S
2 Lausanne-S
3 Oregon-R
4 Oregon-R-c (Df(2)Ore)
5 Stephenville

Chromosome 1 (X)

6 Ax
7 B
8 ct
9 dm/C l, y Hw
10 ec
11 ec ct^6 g/y
12 ec ct^6 v f/y w f:=
13 f B^3/y w f:=
14 f B^3 bb^D/f B^3 + (select)
15 g
16 l(1)7/dl-49, y Hw m^2 g^4
17 sd f B^3/y w f:=
18 w
19 w^a
20 w^{bl}
21 w^{ch}
22 w^{col}
23 w^e
24 w^h
25 w^t
26 w^{48k}(♀ sterile)/y f:=
27 w^{48k28}
28 y g^2 bb
29 y Hw sn^3/y w f:=
30 y pn
31 y sc w m f^5/y f:=
32 y sc w spl
33 y sc w spl m f

34 y sc w^{e} spl
35 y w m f/y w f:=
36 y w sn^{3}
37 y w sn^{3} m B/y w f:=
38 y w $\underline{l}^{48g28}$ sn^{3} bb^{D}/dl-49, sc m^{2} g^{4}
39 y w $\underline{l}^{48g23}$ sn^{3} bb^{D}/dl-49, y Hw m^{2} g^{4}
40 y^{2} ec ct^{6} m f
41 y^{2} ec ct^{6} m f/y w f:=
42 y^{2} sd f car/y w f:=
43 y^{2} w^{a} ec ct^{6} v f
44 y^{2} w^{a} m f

Chromosome 2

45 al
46 b
47 bw
48 bw st
49 c
50 d/Cy(2L), dp^{2} b pr
51 dp
52 dp cn bw
53 dp^{o}
54 L^{2}
55 px
56 S/Cy (E-S)
57 vg
58 vg^{49b2} cn

Chromosome 3

59 Bd^{G}/In(3RC), $\underline{l}$(3)a
60 bx^{34e}/Payne, Dfd ca
61 ca
62 D/Gl
63 e
64 p
65 p^{p}
66 ru h th st p^{p} cu sr e^{s}
67 ru h th st cu sr e^{s} pr ca/T(2,3)Mé
68 ss^{a}
69 st
70 th st cp

Chromosome 4

71 ar/ey^{D}
72 ey

Multichromosomal

73 su^{2}-v, v; bw
74 w$^{48\underline{l}}$; M(3)w/In(3RC), e $\underline{l}$(3)e
75 w^{a}; th st cp
76 y^{SI} sc^{8} In(1)y^{3P}; al^{2} Cy lt^{3} sp^{2}/dp b Pm'; tu h DcxF ca/Sb In(3R).
77 y f:=; bw; e; ci ey^{R}
78 Y^{bb}; cn bw
79 bw; e; ci ey^{R}
80 Cy/Pm; Sb/DcxF

Deficiencies

81 Df(1)w (in In rst^{3}) car/dl-49, y Hw w m^{2} g^{4}
82 y Df(1)w (in In rst^{3}) car/dl-49, y Hw w m^{2} g^{4}
83 Df(1)rst^{2}/dl-49, y Hw m^{2} g^{4}
84 y Df(1)w258-45/dl-49, y Hw m^{2} g^{4}
85 Df(1)w258-48/dl-49, y Hw m^{2} g^{4}

Duplications

86 y f, Dp(1;1)112
87 y w f, Dp(1;1)112/y w f:=
88 y w sn^{3} B, Dp(1;1)112
89 Dp(1;3)N264-58a
90 Dp(1;3)49a7 (Spotted)
91 Dp(1;f?)50k11
92 Dp(1;?)51b7
93 Dp(1;?)51c20

Inversions

94 In(1)Bk, Bk/dl-49, y Hw w m^{2} g^{4}
95 In(1)dl-49, y Hw v m^{2} g^{4}/y w f:=
96 In(1)dl-49, y Hw v m^{2} g^{4}/ClB
97 In(1)dl-49, y Hw w$^{48\underline{l}}$ m^{2} g^{4}/y w f:=
98 In(1)f257-4, y w^{48c22} f257-4
99 In(1)rst^{3}, w rst^{3} f v m/y w f:=
100 In(1)rst^{3}, y rst^{3} bb car/y w f:=
101 In(1)w^{m4}, w^{m4}
102 In(1)w^{m4}, sn w^{m4} (♀ sterile)/y w f:=
103 In(1)w^{m4}, sn w
104 In(1)w^{m4}, y m sn w^{m4}
105 In(1)w^{m4}, y m sn w
106 In(1)w258-52, w258-52 f^{49b15}/dl-49, y Hw w m^{2} g^{4}
107 sc^{S1} B InS w sc^{8}
108 sc^{S1} B InS w^{a} sc^{8} (Muller-5)
109 y$^{(2)}$ B f In(1)dl-49 v sc^{8}
110 y$^{(2)}$ f In(1)dl-49 v w^{a} sc^{8}
111 In(2LR)Gla/Cy
112 In(2LR)Pm/Cy
113 In(3L)Bit/Mé

Translocations

114 T(1;2)Bld/ClB
115 T(1;4)w^{m49a10}, ci
116 T(2;3)Xa/H^{2}

Special Stocks

117 y N264-2/dl-49, y Hw m^{2} g^{4}
118 N264-8/dl-49, y Hw m^{2} g^{4}
119 y N264-32/dl-49, y Hw m^{2} g^{4}
120 y N264-34(Tl, 3L)/dl-49, y Hw m^{2} g^{4}

121 w^{ch} N264-39/dl-49, y Hw m^2 g^4
122 N264-40/dl-49, y Hw m^2 g^4
123 y N264-47/dl-49, y Hw m^2 g^4
124 y N264-53(T1;2L)/dl-49, y Hw m^2 g^4
125 y N264-54/dl-49, y Hw m^2 g^4
126 y N264-58/dl-49, y Hw m^2 g^4
127 y N264-84/dl-49, y Hw m^2 g^4
128 y N264-97/dl-49, Hw g^4
129 y N264-111/dl-49, Hw m^2 g^4
130 N264-118/dl-49, Hw m^2 g^4
131 tumorous-head

ARGENTINA

Buenos Aires: Universidad de Buenos Aires, Instituto de Genetica

Wild Stocks

1 Buenos Aires
2 Mendoza
3 Leningrad

Chromosome 1

4 car
5 ct^{6} g^{2}
6 m
7 ras^{2}
8 w
9 w^{ch}
10 w^{t}
11 w f/dl-49
12 w^{w} f^{5}
13 y f
14 y pn
15 y^{3} w^{a}
16 y w

Chromosome 2

17 Ac at
18 at
19 b at
20 b cn
21 b pr cn vg a sp^{2}
22 dp
23 hy a px sp/Cy sp^{2}

Chromosome 3

24 cd^{42h}
25 D/bal
26 e
27 e^{s} cd ro cmp ca/ Xa ca
28 fll
29 p
30 se^{42g}
31 se fll
32 ss bx
33 st e^{s}

Chromosome 4

34 ci ey^{R}
35 sv
36 sv^{n}

Multichromosomal

37 y; bw; ci ey^{R}
38 w; fll
39 m; at
40 v sc; at
41 w; vg

Translocations

42 T(2;3)Xa ca/Sb

AUSTRALIA

Adelaide, South Australia: University of Adelaide, Waite Agricultural Research Institute

Note: Stocks of Professor D. G. Catcheside, from Cambridge University. The same as listed under Cambridge in DIS-23, p. 45.

Brisbane: The University of Queensland, Department of Zoology

Wild Stocks

1 Oregon-R-C

Chromosome 1

2 w
3 y w^{e}

Chromosome 3

4 e
5 st

Multichromosomal

6 dp; e

Sydney: The University of Sydney, Faculty of Agriculture

Wild Stocks

1 Oregon R-C

Chromosome 1

2 w
3 y w^{e}

Chromosome 2

4 al dp d b c px sp/ Cy sp
5 b j
6 bw

Chromosome 3

7 e
8 e^{s}
9 R/Mé
10 st

Multichromosomal

11 bw; st
12 cb; dp bw
13 dp; e
14 v; bw

Special Stocks

15 Muller inverted stock
16 w^{bf} f^{5}/y v f car (Attached-X)

AUSTRIA

Vienna: Institut f. allgemeine Biologie d. Universität

Note: Stock list unchanged. See DIS-22, p. 40, Zoolog. Institut.

BELGIUM

Brussels: Université Libre de Bruxelles, Laboratoire de Morphologie Animale

Wild Stock

1 Canton-S

Chromosome 1

2 B
3 Df(1)N^8/In
4 f isog.
5 sn^3
6 v
7 w
8 w^{bl} bi B^2
9 w^e bb^1(Y)/ClB
10 y
11 $\underline{y}$
12 y w^a ec cv ct v f/ClB

Chromosome 2

13 dp
14 al dp b lt px
15 Bx
16 bw
17 cn
18 cn bw
19 Df(2)Px^2/Cy L^4 sp
20 L^4
21 M(2)/Cy
22 vg

Chromosome 3

23 e^4
24 se
25 se h
26 se ss
27 ss

Chromosome 4

28 ci ey

Multichromosomal

29 Cy/Pm; Sb/H
30 dp; ss
31 dp; e^4
32 se h; ci ey
33 $\underline{y}$; ci ey

Translocations

34 g^2 B $\underline{XY}^L$ Y^S/$\underline{y}$ Y^S
35 w^mot 5

CANADA

Montreal, Quebec: McGill University, Department of Genetics

Chromosome 1

1 BB/y f
2 car
3 cm
4 dy
5 g
6 m
7 ras
8 rb
9 v
10 w
11 w^e

Chromosome 2

12 b pr c px sp
13 cl
14 cn bw
15 lt stw
16 ltd
17 vg
18 vg bw

Chromosome 3

19 ca
20 cu kar
21 p
22 ru h st p^p ss e^s
23 se h
24 st

Multichromosomal

25 m; bw (1;2)
26 m^D/ClB; bw; st (1;2;3)
27 bw; st (2;3)
28 bw^{59}; st (2;3)
29 bw^{75}; st (2;3)
30 bw^{81}; st (2;3)
31 Cy-RNS/Pm^2; Payne Dfd ca/Sb (2;3)
32 pd; st (2;3)
33 px bw sp; st (2;3)
34 px sp; st (2;3)

FRANCE

Paris: Université de Paris, Laboratoire de Génétique

Note: No new stock list received. See DIS-24, p. 37.

GERMANY

Berlin-Buch: Institut für Medizin und Biologie, Genetische Abteilung

Wild Stocks

1 normal (Berlin wild)
2 normal (England)

Chromosome 1 (X)

3 w
4 w sn^{3}
5 w^{bf}
6 w^{a}
7 w^{e}
8 w^{co} sn^{2}
9 w^{ch} wy
10 w^{m4}
11 gt w^{a}
12 y
13 y^{303}
14 y w
15 y pn
16 y cv v f
17 y w^{a} fa^{n}
18 y w bb
19 y w B bb
20 y fa wy^{2} g^{2}
21 f
22 sc
23 sc rb cv
24 sc ec ct
25 spl
26 m
27 B
28 car bb Y; bb
29 car
30 v
31 cv
32 bb^{2}
33 fa^{n}
34 $\underline{y}$ x +
35 $\underline{y}$ x w^{e}
36 $\underline{y}$ x X^{c2} f
37 $\underline{y}$ x y cv ct c g f B
38 $\underline{y}$ x X^{c}
39 $\underline{y\ w\ f}$ x +
40 $\underline{y\ w\ f}$ x B
41 +/ClB
42 v/ClB
43 w^{e} bb^{1}/ClB
44 y w/ClB
45 y w/y ClB
46 sc ec cv ct^{6} v s^{2} f
car bb^{1}/ClB
47 X^{c}/ClB
48 $\underline{1}$7/dl-49, y Hw w lz^{s}
49 sc^{S1} (InS) w^{a} sc^{8}
50 sc^{S1} B InS w^{a} sc^{8}

Chromosome 2

51 j
52 bw
53 bw^{PP}
54 b cn vg
55 L^{2}/Cy
56 Cy L^{4}/Sb Mé
57 lgl cn bw/Cy cn^{2}
58 fj px sp pb/Mé Sb
59 S Sp ab^{2} ltd/NS px sp
60 5-pl (b pr vg a sp)
61 vg
62 V^{4} Pr/Cy
63 bw cn
64 al dp
65 a sp
66 Cy (Panschin)
67 Cy 2P (Panschin)
68 "F_{1} Cy only"

Chromosome 3

69 e^{11}
70 st
71 p^{p}
72 III-pl (ru h st p^{p}
ss e^{s})
73 ru h st p^{p}
74 Dfd^{r-L}
75 ru h st Dfd p^{p} ss e^{s}
76 ri
77 ss^{a}
78 ss^{a-F}

Chromosome 4

79 ey^{2}
80 ci ey^{R}

Multichromosomal

81 Bld w^{a}/w; Cy
82 w; vg
83 w; vg; e^{11}
84 w; e^{11}
85 cn; ss
86 v; cn
87 v; bw
88 y^{Si} sc^{8} In^{S} y^{3P}; al^{2}
Cy lt^{3} sp^{2}/dp b
Pm^{1}; ru h D InCxF
ca/Sb In3R

39 e, originally CO_{2}-
sensitive (virus)

Berlin-Dahlem: Institut für Genetik der Freien Universität Berlin

Wild Stocks

1 normal (Berlin wild)

Chromosome 1 (X)

2 B
3 cv
4 f
5 m
6 sc ec ct
7 sc ec cv ct^{6} v s^{2} f
car bb^{1}/ClB
8 v
9 w
10 w^{bf}
11 w^{a}
12 w^{co} sn^{2}
13 w^{e-3}
14 w^{ch} wy
15 w sn^{3}
16 w y
17 y cv v f
18 y w
19 +/ClB

Chromosome 2

20 b cn vg
21 bw
22 Cy (Panschin)
23 L^{2}/Cy
24 S Sp ab^{2} $\underline{l}$td/NS
px sp
25 vg

Chromosome 3

26 e^{11}
27 ri

Chromosome 4

28 ci/ey^{R}
29 ey^{2}

Multichromosomal

30 cn; ss
31 e; vg
32 y^{Si} sc^{8} InS y^{3P}; al^{2} Cy lt^{3} sp^{2}/dp b Pm^{1}; ru h D InCxF ca/ Sb In3R

Virus?

33 e CO_2-sensitive (l'Héritier)

Göttingen: Max Planck-Gesellschaft, Institut für Tierzucht und Tierernährung

Chromosome 1

1 br ec rb
2 ClB/+
3 gt w^{a}
4 sc^{8} B
5 sc rb cv
6 sc^{8} Y/y f x sc^{8} Y/X^{c2} y v
7 w^{bf}
8 w^{ch} wy
9 w^{co} sn^{2}
10 w^{co} v f
11 w^{e3}
12 w sn^{3} B
13 y ac v bb
14 y pn
15 y w bb
16 y w cv v f
17 y^{2} sc^{8} w^{a} cv v f

Chromosome 2

18 a sp^{2}
19 b pr vg a sp
20 cn bw
21 fj px sp
22 L^{2}/Cy
23 L Cy/ds cg
24 lgl cn bw/Cy cn^{2}

Chromosome 3

25 Dfd^{r-L}
26 Gl Sb/LVM
27 pb/C Mé Sb C
28 ri
29 ru h st Dfd^{r-L} p^{p} ss e^{s}
30 ss^{a}
31 ss^{a-F}

Multichromosomal

32 ant; ro

Attached-X

33 y w f
34 y x kz g^{2} B
35 y v f x fa^{no}

Göttingen: Zoologisches Institut der Universität

Wild Stocks

1 Berlin
2 Bovenden
3 Oregon-Dresden

Chromosome 1

4 B
5 ClB/+
6 car bb; Y^{bb}
7 fa^{n}
8 fa^{no}
9 m
10 sc^{1}
11 sc^{1} sc ct^{6}
12 sc^{S1} B InS w^{a} sc^{8}
13 v M(1)n/In(1)AM
14 w
15 w^{a}
16 w^{m}
17 w sn^{3}
18 y
19 y cv v f
20 y cv v f car

Chromosome 2

21 al
22 al dp
23 dp
24 fes lt/Cy al^{2} lt^{3} L^{4} sp^{2}
25 j
26 L^{2}/Cy
27 vg

Chromosome 3

28 e^{11}

Chromosome 4

29 ci^{D}/ey^{D}
30 ey^{2}

Multichromosomal

31 cn; ss
32 Cy al^{2} lt^{3} L^{4} sp^{2}/+; C Mé Sb C/+
33 fj px sp; pb/C Mé Sb C
34 vg; e^{11}
35 w; j; e^{11}; ey^{2}
36 y; dp; se; ey^{2}
37 Cy/Pm ds^{33k}; H/C Sb

Attached-X

38 y

Hamburg-Eppendorf: Universitäts-Frauenklinik, Strahlenbiologische Abteilung

Wild Stock

1 normal (Berlin wild)

Chromosome 1 (X)

2 ClB/+
3 sc^{S1} B InS w^{a} sc^{8}
4 sc^{8} Y/y f x sc^{8} Y/X^{c2} y v
5 w
6 X^{c}/ClB
7 y

Multichromosomal

8 cn; ss

Heidelberg: Zoologisches Institut der Universität

Wild Stocks

1 Berlin-normal
2 Canton-S
3 Ore-R-C

Chromosome 1

4 B
5 ClB/+
6 fa^n
7 sc (?)
8 tw
9 tw/y Hw
10 w
11 w sn^3
12 w^{bf}
13 w^m (?)
14 X^{c2}
15 y w f/+
16 y/+

Chromosome 2

17 dp
18 ri
19 vg

Chromosome 3

20 e^{11}
21 In(3R)bxd^{101}/sbd^2 bx^3
22 se
23 ss^{a-F}

Chromosome 4

24 ar/ey^D

Multichromosomal

25 vg; e^{11}

Neustadt, Schwarzwald: Hirnforschungsinstitut

Note: Stocks previously listed here have been transferred to the Max Planck-Institut für Tierzucht und Tierernährung, Bunsenstrasse, Göttingen. (See Göttingen) Some deficiencies of the X chromosome that occurred in the progeny of urethane-treated males are still kept in Neustadt.

GREAT BRITAIN

Bayfordbury, Hertford, England: John Innes Horticultural Institution

Wild Stocks

1 Amherst-3
2 Coombe Hill
3 Crimea
4 Ealing
5 Florida-4
6 Great Braxted
7 Hampton Hill
8 Merton Park
9 Ockley
10 Oregon-R (inbred 272 generations)
11 Rothamsted
12 Samarkand
13 Samarkand (inbred 260 generations)

Chromosome 1

14 aw^1/y Hw m^2 g^4, dl-49) non-allelic
15 aw^2/y Hw m^2 g^4, dl-49)
16 B
17 B (inbred for 30 generations)
18 BB
19 crm/Muller-5
20 ct v g
21 cv ct v
22 f B^iB^i
23 Lg/y Hw m^2 g^4, dl-49
24 rab
25 rb^{50j}
26 sc ec cv
27 sc ec cv ct v g f/y Hw m^2 g^4 dl-49
28 sc bz^{50j}/Muller-5
29 sc v r^P/y Hw m^2 g^4, dl-49
30 shf-ov/y Hw m^2 g^4, dl-49
31 sml/y Hw m^2 g^4, dl-49
32 sy Bx/sc^{S1}
33 v
34 v g f
35 vs^{P1}
36 w
37 w^P
38 w vs bz v g un/Muller-5
39 y fa wy^2 g^4
40 y w

Chromosome 2

41 al dp b pr c px sp/Cy
42 b pr vg
43 S Sp al^2 ltd/NS px sp

Chromosome 3

44 H^{P1}/Sb
45 H^{P2}/Sb
46 Mc/Sb
47 Mé ca/rucuca

Chromosome 4

48 ci ey^R

Attached-X

49 f(Y^S) x X Y^L(Y^S)
50 y (isogenic with Samarkand inbred)
51 y x fu^g
52 y v f car x y sc v f car

Multichromosomal

53 ClB; Cy L^4/Pm; H/Sb
54 dp; e

Inversions

55 ClB/sc v f car
56 Muller-5
57 Muller-5 (isogenic with Samarkand inbred)
58 N^P/+
59 rg^P v (previously listed as fas)
60 y fa^n dl-49
61 y Hw m^2 g^4 dl-49/dow
62 y rst^3 car bb

Duplications

63 Bt/y Hw m^2 g^4, dl-49

Translocations

64 T(1;3R) fd/y Hw m^2 g^4, dl-49 (with inversion in 3R)
65 T(1;2L) vs^{P4}/Muller-5

Birmingham, England: The University, Department of Genetics

Wild Stocks

1-16 wild types

Inbred Stocks, etc.

17 Oregon (375 gen)
18 Samarkand (175 gen)
19 f x Oregon (14 back x)
20 f x Samarkand (14 back x)

Chromosome 1

21 f B^iB^i
22 m
23 w m
24 w m B
25 w^a v

Chromosome 2

26 c
27 c px sp
28 mr bs^2/Cy Sp^2
29 L^4
30 S Sp ab^2 ltd/NS px sp
31 al Cy L^4 sp/Pm with Oregon
32 al Cy L^4 sp/Pm with Samarkand

Chromosome 3

33 cu
34 st
35 st p^P

Multichromosomal

36 ClB; al Cy L^4 sp/Pm; H/sb
37 v; bw
38 vg; st p^P
39 w v; vg sp
40 y; ey^2

Edinburgh, Scotland: Edinburgh University, Institute of Animal Genetics

Wild Stocks

1 Bonnyrigg
2 Canton-S
3 Crianlarich
4 Eket
5 Hendon A
6 Hendon B
7 Ibadan
8 Ischia
9 Kaduna (N. Nigeria)
10 Kumba (Br. Cameroons)
11 Nettlebed
12 Oregon-K
13 Oregon-R
14 Oregon-S
15 Refrew
16 Samarkand
17 Urbana-S
18 Victoria
19 Wild Edinburgh
20 Oregon-K inbred
21 Oregon-R inbred
22 Samarkand inbred

Chromosome 1

23 B
24 bi ct^{6} v
25 car bb; y^{bb}
26 ClB/fu
27 ClB/or
28 ClB/sc ct m f car
29 ClB/sc ct v f car
30 ClB/sc v f car (scar)
31 ClB/y w lz
32 beb/y Hw m^{2} g^{4}
33 dow/or
34 dow/y Hw m^{2} g^{4}, delta-49
35 fw
36 sc ec cv ct^{6}
37 ct^{6} v g
38 v g f
39 sc ec cv ct^{6} v g^{2} f/sc^{S1}(InS)w^{a} ct sc^{8}
40 sc^{S1}(InS)wa ct sc^{8}
41 sc^{S1} B InS w^{a} sc^{8} (Muller-5)
42 sc^{8} B/w lz^{s}, delta-49
43 w
44 w^{a}
45 w^{co} v
46 w^{m4}
47 w sn^{3} B
48 y fa^{n}, delta-49
49 y fa wy^{2} g^{4}
50 y sc v f car
51 y w
52 y w sn^{3}
53 v

Attached-X

54 Muller-5 and w
55 Muller-5 and w lz^{s}, delta-49
56 Muller-5 and y Hw m^{2} g^{4}, delta-49
57 Muller-5 and y sc v f car
58 y and fu^{gl}
59 y and y^{2} sc^{8} w^{a} B
60 y and y w lz
61 y v f and sc^{S1}(InS)w^{a} sc^{8}
62 y w f and w

Closed-X

63 X^{c1} y/sc^{S1}
64 X^{c2} cv v f/sc^{S1}
65 X^{c2} y v and y
66 y v f car/X^{c2} car^{+} (small ring) and v wy f car/X^{c2} car^{+} (small ring)

Deletions and Duplications

67 ct^{-}/sc^{S1}(InS)w^{a} ct sc^{8}
68 Df(3) Bxl/Xa T(2;3)
69 y^{-} sc^{S1}(InS)wa ct sc^{8}/y w lz
70 y w f.Dp. sc^{S1}
71 y w f.Dp. sc^{S1} and y w f

Chromosome Y

72 f. Y^{s}/Y^{L}
73 f. Y^{s}/sc Y^{L} and y w f/sc Y^{L}
74 X Y^{L}/Y^{s} (Neuhaus)
75 y v f Y^{L} and f/Y^{s}
76 T(1;4) B^{s}, Y^{s}/sc Y^{L} and y w f sc Y^{L}

Chromosome 2

77 al dp b pr c px sp (all)
78 b blt
79 b rn px/Cy pr
80 b cn vg
81 Bl L^{2}/Cy
82 Cy/Pm
83 d b l/Cy pr
84 dp
85 ds b pr cg/Cy dp
86 ds ap^{4} vg/Cy al^{2} L^{4} sp^{2}
87 lf al/Cy L^{4} sp
88 nw^{2}/Cy- RNS
89 oph
90 Pin
91 vg

Chromosome 3

92 Tp(3)bxd^{100} ri/T(2;3)Mé
93 bxd e^{s}/sbd^{2} bx^{3}
94 Dfd/th st p^{b} p^{p} ss bx
95 e
96 Gl/LVM
97 Gl Sb/LVM
98 Ly/D
99 Mé/H
100 rucuca
101 ru h th st cu sr e^{s} Pr ca/T(2;3)Mé
102 Sb/LVM
103 ss
104 ss^{a-B}
105 ss^{a-Sn} B
106 ss^{a-Sn} B/T(2;3)Mé
107 ss^{a-40a}
108 ss e

Chromosome 4

109 ci ey^{R}
110 ci^{D}/ey^{D}
111 ey^{2}
112 ey^{4}
113 ci gvl ey^{R} svn

Multichromosomal

114 ant; ro
115 C1B; Cy al^2 sp^2; T(2;3)Me
116 C1B; Pm; H
117 Cy/Bl L^2; D/LVM
118 Cy/Pm; Ly/D
119 dp; e
120 Muller-5; dp; e
121 sc^{S1} and y; bw; e; ey
122 sc^{S1}(InS)w^a sc^8; dp; e
123 sc^{S1}(InS)w^a sc^8 and y v f; dp; e
124 v; bw^{Va}/Vl L^2
125 v and y v f; bw^{Va}/Bl L^2
126 w; vg

Translocations

127 e^s cd ro cmp ca/Xa ca
128 T(1;3) and y; bw; e; ey
129 T(2;3); Cy; e

Triploids

130 y/Muller-5 ct

Inversions

131 In(2LR)40d/Cy sp^2
132 In(3R)ss^{101} In(3R)Payne/Sb Bxl
133 D^3 Sb ca^2/Payne
134 cmp ca/In(3R)ce

Glasgow, Scotland: University of Glasgow, Department of Genetics

Wild Stocks

1 Florida-4 (inbred)
2 Oregon-K (inbred)

Chromosome 1

3 B
4 cv ct v
5 C1B/y w lz
6 sc ec cv
7 sn^3
8 v
9 w
10 w^{FF33}
11 w^a
12 w^{bf2}
13 w^{bl}
14 w^{co}
15 w^h
16 w^i
17 w^{m4}
18 w^P
19 w^{sat}
20 w^t
21 w m f
22 w sn^3
23 w sn^3 B
24 y w
25 z w^{1P}
26 w z^{11E4}
27 z w^{14G3}

Attached-X

28 y/y w^a
29 y/sc z w^{17G2} ec ct
30 y v f/
31 y v f/sc ec cv ct
32 y v f/r^{50f}
33 y v f/w^{FF33}
34 y w f/z w^{2P}
35 y w f/z w^{1G1} ct

Chromosome 2

36 b cn vg
37 bw
38 dp
39 fj px sp

Chromosome 3

40 e

Multichromosomal

41 y v f/v; bw^{VA}/Bl L^2
42 bw e
43 y v f/w^{FF37} ex

Harpenden, Herts, England: Rothamsted Experimental Station

Rothamsted wild-type
Merton Park
Kenya
Rothamsted x Oregon x Samarkand x Crimea--in addition to parent stocks

London, England: University of London, King's College, Department of Botany

Note: Only stocks not included in the Edinburgh stock list in DIS-24 are listed below.

Wild Stocks

1 Oregon-R (inbred)
2 Samarkand (inbred)

Chromosome 1

3 B
4 f $B^{i}B^{i}$
5 m^{49c}
6 m/f B
7 na/Muller-5
8 w^{50h}/Muller-5
9 w m f/sc^{S1} B InS w^{a} sc^{8}

Chromosome 2

10 b pr vg
11 Bl/Cy, al^{2} lt^{3} L^{4} sp^{2} cn^{2}
12 cn bw
13 cn vg bw

Chromosome 3

14 ro e^{S} cd cmp ca/Mé, In(3R)C Sb e $\underline{l}$(3)e

Chromosome 4

15 cat/ci^{D}

Multichromosomal

16 bw; st
17 Cy al L^{4} sp/Pm; H/Sb sr In(3R)Mo
18 v; bw

London, England: University College, Department of Biometry

Wild Stock

1 Oregon +

Chromosome 1

2 Kenya In
3 Kenya In w
4 w^{bl}
5 w^{bl} wy f
6 w^{e-z}
7 w^{a} m
8 w^{ch} wy
9 w^{co} st bristle
10 w^{n4}
11 bsd
12 v
13 Bx^{3}
14 sd
15 BB
16 v sd
17 y m car
18 In rst^{3} rst^{3}
19 S.I.
20 $\underline{y}$/w^{a} m f
21 $\underline{y}$/rb ct m In
22 $\underline{y}$/y rst^{3} m car
23 C$\underline{l}$B/sc vf car
24 N^{8}/+
25 N^{8}/dl-49 y Hw m^{2} g^{4}

Chromosome 2

26 bw
27 pk bw
28 cn bw
29 cn

Chromosome 3

30 ss
31 e ss^{a}
32 ve ss^{a}
33 ss^{a}
34 st
35 ve

Multichromosomal

36 v bw
37 dp ss^{a}
38 Mc/Sb
39 vr^{x} (sd)

ITALY

Milano: Università di Milano, Istituto di Genetica

Wild Stocks

1 Aspra
2 Crkwenika (Oregonized except Y)
3 Gaiano
4 Luino (Oregonized except Y)
5 Moltrasio
6 Oerlikon (Oregonized except Y)
7 Oregon-R
8 S.Maria
9 Valdagno
10 Varese

Chromosome 1

11 B
12 Bx^{3} (Oregonized)
13 fa^{n}
14 m^{2} g^{4}
15 N^{B-S}
16 sd
17 w
18 w^{a}
19 w^{bl}
20 w^{bf}
21 w^{e}
22 y Hw

Chromosome 2

23 b cn vg

24 blt
25 blt^{S}
26 bw^{5}/Cy
27 cn
28 ll
29 so
30 so^{2} b cn
31 so^{C}
32 spt

Chromosome 3

33 cp
34 gl^{3}
35 obt
36 ri-se ss k e^{s} ro
37 rs^{2}

Not Localized

38 abab^{49} cn (autosomal)

Inversions

Chromosome 1

39 ClB/+
40 ClB y/y Hw m^{2} g^{4}
41 l(1)7/dl-49 y Hw m^{2} g^{4}

Chromosome 2

42 Cy sp/Pm

Chromosome 3

43 H/Sb sr In(3R)Mé

Chromosomes 2 and 3

44 L^{4} Cy sp/Pm; H/Sb sr In(3R)Mé

Pavia: Università, Istituto di Genetica

Wild Stocks

1 Ankara
2 Belluno
3 Chieti
4 Crkvenica
5 Jaslo
6 Oregon-R
7 Suna

Inbred Stocks

8 Oregon (274 gen.)
9 Samarkand (162 gen.)

Chromosome 1

10 B
11 ClB/+
12 Df(1)N8/y Hw m^{2} g^{4}, dl-49
13 lz/ClB
14 sc ct v f car/ClB
15 sc^{S1} B InS w sc^{8}
16 sc^{S1} B InS w^{a} sc^{8}
17 sc^{S1} InS w^{a} ct sc^{8}
18 t
19 v^{m}
20 w
21 w^{a}
22 w B
23 w^{Berlin}
24 w^{bf}
25 w^{co} sn
26 w^{e}
27 w^{i}
28 w^{m-4}
29 w^{m-4000}
30 w^{m-6000}
31 w^{t}
32 y/+
33 y w^{a} cv v f
34 y w sn

Chromosome 2

35 b cn vg
36 b pr e px sp
37 Cy/L^{2}
38 oo
39 vi

Chromosome 3

40 app
41 D Sb/e CL-CR
42 Dfd/Cx D
43 Dl^{7}/In(3R)Mé Sb sr
44 fj-s
45 Gl Sb/LVM
46 ri-s se ss k e^{s} ro
47 rs^{2}
48 sc h th
49 ss^{a-44-a}
50 st
51 tt wo

Chromosome 4

52 ey^{2}
53 sv

Chromosome Y

54 f. Y^{S}/sc Y^{L} and
y w f/sc Y^{L}

Multichromosomal

55 al L^{4} Cy sp/Pm; H/Sb sr
In(3R)Mé
56 bw; e^{4} wo ro
57 cn; ss
58 e^{11}; st ss
59 px^{43j} oo; ru jv se st ca

Special stocks (isogenic)

60 cn; ss

JAPAN

Kyoto: Kyoto University, Zoological Institute

Wild Stocks

1 Canton-S
2 Miyazaki (Japan)
3 Oregon
4 Oregon-R-S
5 Stephenville
6 Tokyo (Japan)

Chromosome 1

7 B
8 B (from Berkeley)
9 BB
10 B(bb)
11 Bx (from Berkeley)
12 Bx^{50a}
13 ec ct g^{2} bb^{1}/C$\underline{1}$B
14 f B^{i}B^{i}/$\underline{y}$
15 fa
16 f
17 fu^{g}/$\underline{\text{y v f car}}$
18 g^{2} ty/$\underline{y}$
19 lz^{3}/y f:=
20 m
21 N
22 rst^{2}/dl-49, y Hw m^{2} g^{4}
23 sc^{S1} B InS w^{a} sc^{8} (Muller-5)
24 sd mc
25 v
26 w
27 w B
28 w B^{i}
29 w m
30 w f
31 w^{a}
32 w^{e}
33 y
34 y ac v
35 y w
36 y w f

Chromosome 2

37 al dp b pr c px sp/Cy
38 al dp b pr c px sp/Cy pr
39 ap^{4}/Cy
40 b (Inversion ?)
41 b gp
42 bw
43 bw/Cy
44 bw dp
45 bw vg
46 cn
47 dp
48 dp
49 dp bw
50 dp vg
51 L^{2}
52 M(2)173/Cy al^{2} lt^{3} L^{4} sp^{2}
53 px
54 S Sp ab^{2} lt d/NS, px ap
55 S/Cy E-S
56 vg
57 vg^{no}
58 vg^{ni}
59 vg^{np}
60 vg^{nG}
61 vg^{Nw} Hia/T(2;3)S^{M} Cy

Chromosome 3

62 Bx 1/Payne, Dfd ca
63 cu
64 DL^{3}/In(3R)C, e
65 e^{11}
66 Gl Sb/LVM
67 H^{2}/Xa
68 M(3)w/In(3R)C, e l(3)e
69 p^{p} bx sr l^{s}
70 ro
71 ru
72 ru h th st cu sr e^{s} pr ca/T(2;3)Mé
73 ru h th st cu sr e^{s} ca/Mé Lx
74 (Sbd^{2}) bx^{3} Bx^{L}/Xa
75 Sbd^{2} bx^{3}/Bx^{L}
76 se
77 se ss ro
78 st
79 ve

Chromosome 4

80 ci ey^{R}
81 ey
82 gvl ey^{R}
83 Scn/ey^{D}
84 svn

Multichromosomal

85 cn; bw
86 dp; se
87 S/Cy; D/Cx
88 st; bw
89 v; bw
90 w^{e} f; sr
91 vg; se
92 y; bw; e; ci ey^{R}

Translocations

93 T(2;4)d/Cy, pr
94 T(2;4)A 29/Cy

Special Stocks

95 w258-11 (Df w)	y w258-11/dl-49, y Hw m^2
96 w258-14 (Df w)	y w258-14/dl-49, y Hw m^2 g^4
97 w258-42 (Df w)	y w258-42/dl-49, y Hw m^2 g^4
98 w258-45 (Df w)	y w258-45/dl-49, y Hw m^2 g^4
99 N264-72 (Df)	y N264-72/dl-49, y Hw m^2 g^4
100 N264-88 (Df)	N264-88/dl-49, y Hw m^2 g^4
101 N264-112 (In)	y N/dl-49, Hw m^2 g^4
102 ct268-2b	ct268-2/dl-49, y Hw m^2 g^4
103 N264-8	N264-8/dl-49, y Hw m^2 g^4

Unanalyzed

104 Virus? CO$_2$-sensitive e (l'Héritier)

Misima, Sizuoka-Ken: National Institute of Genetics

Wild Stocks

1 Canton-S
2 Misima
3 Oregon-R-S
4 Oregon-2
5 Stephenville
6 Tokyo

Chromosome 1 (X)

7 B
8 BB
9 bb Y^{bb}
10 lz^3/y f
11 m
12 sd mc
13 v
14 w B
15 w f
16 w^a
17 y ac v
18 y f w
19 y w

Chromosome 2

20 al dp b pr c px sp/Cy pr (all).
21 ap^4/Cy
22 b arch l(2)C/Cy, L^4 sp^2
23 bw/Cy
24 bw dp
25 cn
26 dp^x
27 pr en
28 S/Cy E-S
29 S Sp ab^2 ltd/NS, px sp
30 Str 3104
31 vg
32 vg^{ni}
33 vg^{no}
34 vg^{nG}
35 vg^{nw}/S^M Cy

Chromosome 3

36 bx/Payne Dfd ca
37 cu
38 e^{11}
39 Gl Sb/LVM
40 (sbd^2) bx^3 Bxl/Xa
41 ru h th st cu sr e^s Pr ca/Me LX
42 st

Chromosome 4

43 ey
44 gvl ey^R

Multichromosomal

45 S/Cy; D/cx
46 v bw
47 g^2/Cy y

Closed-X

48 X^c, y/fB

Inversion

49 sc^{S1} B InS w^a sc^8 (Muller-5)

Special Stocks

50 w258-11 (Df w)	y w258-11/dl-49, y Hw m^2
51 w258-14 (Df w)	y w258-14/dl-49, y Hw m^2 g^4
52 w258-45 (Df w)	y w258-45/dl-49, y Hw m^2 g^4
53 N264-88 (Df)	N264-88/dl-49, y Hw m^2 g^4
54 ct268-2b	ct268-2/dl-49, y Hw m^2 g^4
55 N264-8	N264-8/dl-49, y Hw m^2 g^4

Unanalyzed

56 Virus? CO2-sensitive (l'Héritier)

Osaka: Osaka University, Faculty of Medicine, Genetical Laboratory

Wild Stocks

1 Oregon
2 Oregon-R-S
3 Stephenville
4-20 Wild types in Japan

Chromosome 1

21 B
22 BB
23 B bb
24 Bx^{50a}
25 Df(1)N264-8/dl-49 y Hw m^2 g^4
26 ec ct g^2 bb^1/C$\underline{l}$B
27 f
28 v
29 w
30 w B^1
31 w mt
32 w^a
33 w^e
34 y ac v
35 y w

Chromosome 2

36 bw
37 bw/Cy
38 cn
39 cn bw
40 dp
41 px
42 vg
43 al dp b pr c px sp/Cy

Chromosome 3

44 e^{11}
45 ro
46 ru
47 se
48 st

Chromosome 4

49 ey

Multichromosomal

50 v; bw
51 w^e f; sr

Sapporo: Hokkaido University, Institute of Zoology

Wild Stocks

1 Oregon-R-C
2 Oregon-R-S
3 Otaru
4 Sapporo
5 Shioya
6 Utsunomiya

Tokyo: Tokyo Metropolitan University, Department of Biology

Wild Stocks

1 Canton-Special
2 Okinawa
3 Omogô, Shikoku
4 Oregon
5 Senzoku, Tokyo
6 Tokyo

Chromosome 1

7 B
8 B (Calif. U.)
9 BB
10 bb Y^{bb}
11 Bx (Calif. U.)
12 Bx^3
13 Bx^{50a}
14 ec ct^6 g^2 $bb^{\underline{l}}$/C$\underline{l}$B
15 f B^iB^i/$\underline{y}$
16 fu^g/$\underline{y\ v\ f\ car}$
17 $fw^{50\underline{l}}$
18 lz^3/y f:=
19 m
20 $N^{49\underline{l}}$/dl-49, y Hw m^2 g^4
21 rst^2/dl-49, y Hw m^2 g^4
22 sd mc
23 sw
24 v
25 v^{51a10}
26 v^{51b25}
27 w
28 w B
29 w f
30 w m
31 w^a
32 y
33 y w
34 y w f

Chromosome 2

35 al dp b pr c px sp/Cy pr (all)
36 al dp b pr c px sp/Cy
37 ap^4/Cy
38 b (inversion?)
39 b^{51i18}
40 bw/Cy
41 bw dp
42 bw vg
43 cn
44 Cy/$\underline{l}$(2)50(Nozawa)
45 Cy/$\underline{l}$(2)50(Nozawa) (selected 16 gen.)
46 Cy/abnormal lethal (Yoshida)
47 dp^x
48 L^2
49 M(2)173/Cy al^2 lt^3 L^4 sp^2
50 pr en
51 S Sp ab^2 ltd/NS, px sp
52 Str 3104
53 vg
54 vg^{no}
55 vg^{ni}
56 vg^{np}
57 vg^{nG}
58 vg^{Nw} Hia/T(2;3)S^M Cy

Chromosome 3

59 $Bd^{49\underline{l}}$/H
60 $Bd^{49\underline{l}}$/Sb
61 Bxl/Payne, Dfd ca
62 cu
63 Dl^3/In(3R)C, e
64 $Dl^{49\underline{l}}$/$\underline{l}$(3)
65 e^{11}
66 e^{51g}
67 Gl Sb/LVM
68 H^2/Xa
69 H^{51c}/Sb
70 Ly/D^3
71 M(3)w/In(3R)C, e $\underline{l}$(3)e
72 $P^{\overline{P}}$ bx sr e^s

73 ru h th st cu sr e^s Pr ca/
T(2;3) Mé
74 ru h th st cu sr e^s ca/Mé LX
75 Sb/$\underline{1}$(3) (dominigene for yg)
76 (sbd^2) bx^3 Bxl/Xa
77 sbd^2 bx^3/Bxl
78 se ss ro
79 st
80 ve
81 N-X/In(3R), $\underline{1}$(3R)
82 N-X ss ro/Dl^{49}$\underline{1}$
83 N-X bx sr e^s/bx sr e^s

Chromosome 4

84 bt
85 ci ey^R
86 ey
87 gvl ey^R
88 Scn/ey^D
89 sv^n

Chromosome Undetermined

90 crossvein broken
91 eye mutant-1
92 eye mutant-2
93 (ro)
94 tammo
95 (gp)

Multichromosomal

96 Cy/Pm, ds^{33k} (dp,b); H/C Sb
97 Cy/Pm; PP bx sr e^s
98 S/Cy; D/Cx (2;3)
99 vg/Cy; M(3)w/In(3), $\underline{1}$(3)
100 vg^{no}/vg^{no}; M(3)w/In(3), $\underline{1}$(3)
101 vg; se
102 w^{50k}; N-X In(3)/$\underline{1}$(3), In(3)

Inversion

103 sc^{S1} B InS w^a sc^8 (Müller-5)

Translocations

104 T(2;4)d/Cy, pr
105 T(2;4)A29/Cy

Special Stocks

106 w258-11 (Df w)	y w258-11/dl-49, y Hw m^2
107 w258-14 (Df w)	y w258-14/dl-49, y Hw m^2 g^4
108 w258-42 (Df w)	y w258-42/dl-49, y Hw m^2 g^4
109 w258-45 (Df w)	y w258-45/dl-49, y Hw m^2 g^4
110 w264-72 (Df)	y N264-72/dl-49, y Hw m^2 g^4
111 N264-88 (Df)	N264-88/dl-49, y Hw m^2 g^4
112 N264-112 (In)	y N/dl-49, y Hw m^2 g^4
113 ct268-2b	ct268-2/dl-49, y Hw m^2 g^4
114 N264-8	N264-8/dl-49, y Hw m^2 g^4

Unanalyzed

115 abnormal wing
116 intensifier of Bd
117 modifier of vg^{no}

Virus?

118 CO_2-sensitive e (l'Héritier)

Tokyo: University of Tokyo, Botanical Institute
Laboratory of Genetics

Wild Stocks

1 Tokyo
2 Oregon

Chromosome 1

3 B
4 v
5 w

Chromosome 2

6 bw/Cy
7 vg

Chromosome 3

8 cu

NETHERLANDS

Utrecht: Zoölogisch Laboratorium der Rijksuniversiteit

Note: Only stocks not represented at Edinburgh are listed.

Wild Stocks

1 Berlin Inzucht
2 Utrecht

Chromosome 1

3 lne f/sc^{S1}(InS) w^a sc^8
4 w m f
5 y w^a cv v f

Chromosome 2

6 Bl L/Cy
7 Cy al^2 lt^3 L^4 sp^2/Pm
8 dp^{50c}

9 fes lt/Cy al^{2} lt^{3} L^{4} sp^{2}
10 J/In(2L)+, 1(2)B
11 J^{34e}

Chromosome 3

12 Cx, D/In(3R)Mé Sb
13 De/e
14 D Sb/e CL + CR
15 D^{3} H/Payne
16 Dl/In(3R)C 1(3)a
17 ltr/e In(3R) In(3L)
18 ltr/Mé Sb

Chromosome 4

19 ey
20 ey^{4}

Multichromosomal

21 Cy al^{2} lt^{3} L^{4} sp^{2}/Pm; Cx, D/In(3R)Mé Sb
22 e/e; ey^{D}/+
23 lgl; ltr/T(2;3)E

NORWAY

Oslo: Universitetet, Arvelighetsinstituttet

Wild Stocks

1 Florida
2 Oregon

Chromosome 1

3 B
4 B"/y
5 bb
6 br ec rb
7 Bx^{3}
8 Bx^{r49k}
9 car
10 car px-like
11 ec ct^{6} v g^{3}/C1B
12 f
13 fa
14 fu^{g}/y v f car
15 g f B/y
16 g^{37c}
17 g^{37f}
18 gt w^{a}/gt bb^{11}
19 lz/y
20 lz^{51d10}/y
21 m
22 na/sc^{8} dl-49 B w^{a} lz^{s}
23 od Bx^{r49k}
24 od car
25 pn
26 sc cv lz/y
27 sc cv v f
28 sn^{2} B/y
29 sw
30 w
31 w m f
31a w^{50j4}
32 w^{a}
33 w^{ch} wy
34 y
35 y ec ct c f
36 y sc w^{e} ec rb
37 y pn
38 y w

Chromosome 2

39 a px or
40 al dp b pr c px sp
41 al dp b pr c px sp/Cy, pr
42 b j pr cn
43 b pr ad
44 bw
45 bw mt^{A} vg
46 cg c/U
47 dp
48 dp^{51f26}
49 dp^{T}/In(2R)S^{2} E-S
50 ds ft dp^{V2} 1(2)M b pr/Cy, dp^{2}
51 ds S G b pr/Cy, L
52 fj px sp
53 ho
54 j^{49j18}
55 j^{50e5}
56 L^{2}/Cy
57 L^{5}
58 lt std/Cy, sp^{2}
59 M(2)l^{2}/Cy, L^{4} sp^{2}
60 M(2)z/In(2L)t, 1(2R)
61 M(2)50j7/Cy, L^{4}
62 mr bs^{2}/Cy, sp^{2}
63 ms/Cy
64 pr en
65 pr^{41}
66 rl
67 S/NS px sp
68 S^{37b}/Cy
69 sca
70 stw^{D}
71 tu(2)49k
72 vg

Chromosome 3

73 cp
74 cu kar
75 cv-c
76 D^{3} H/Payne
77 Dl^{K}/Sb
78 dv/Mé
79 e^{4} wo ro
80 gl^{3}
81 Gl Sb/LVM
82 Hn^{r} h
83 jv dv h/In(3LR), Dfd
84 jv Hn^{r} h
85 jv se
86 Ly/D^{3}
87 M(3)bb/LVM
88 M(3)w/In(3R)C, e 1(3)e
89 ma fl
90 ma^{49d}
91 p^{42a}
92 Pr/In(3R)C, e
93 ra
94 ri
95 rs^{2}
96 ru dv h/In(3LR)P, Dfd
97 ru h th st cu sr e^{s} ca
98 se
99 se ss k e^{s} ro
100 st
101 st e^{2}
102 tt wo
103 ve
104 W

Chromosome 4

105 ar ey^{D}
106 bt
107 ci ey^{R}
108 ey^{2}
109 sv^{2}

Multichromosomal

110 Cy/Pm; H/C Sb
111 y; bw; e; ci ey^{R}

Closed-X

112 X^{c}, y/f B

Deficiencies

113 Df(2)MS^{10}/Cy pr
114 Df(2)rl^{10a} lt cn/Cy

Translocations

115 T(2;3)Cy
116 T(Y;3)

Note: Dr. Vilhelm Kiil's stock lists for both melanogaster and funebris are unchanged.

SOUTH AFRICA

Johannesburg: University of the Witwatersrand, Department of Zoology

Wild Stocks

1 Canton-S
2 Cedara
3 Florida
4 Graaff Reinet
5 Johannesburg
6 Lourenco Marques
7 Mowbray
8 Nelspruit
9 Stellenbosch
10 West Rand
11 Zoutpansberg

Chromosome 1

12 bi ct^{6} g^{2}
13 bo
14 B
15 car
16 cm
17 cm car
18 cm g^{3} car
19 ct v
20 ct v dy g
21 ct^{6}
22 ec
23 ec ct^{6} v g^{3}
24 ec ct^{6} v g^{3}/ClB
25 f
26 f B
27 f B/y
28 f^{5}
29 f^{5} m
30 f^{5} v
31 g
32 g^{2}
33 g^{3}
34 g^{5} car
35 m
36 m f
37 pn^{2}
38 ras dy
39 ras^{2}
40 ras^{3} m
41 rb
42 rb car
43 rb cm g^{3}
44 rb cm car
45 rb cx
46 rb g^{3}
47 rb g^{3} car
48 svr w^{a}
49 v
50 v^{36f}
51 v g^{3}
52 w
53 w m
54 w m f
55 w^{a}
56 w^{a3}
57 w^{a4}
58 w^{a} rb
59 w^{bl}
60 w^{bl} rb
61 w^{ch}
62 w^{ch} rb
63 w^{ch} wy
64 w^{co}
65 w^{co} rb
66 w^{col}
67 w^{e}
68 w^{e2}
69 w^{e} car
70 w^{e} cm
71 w^{e} g^{3}
72 w^{e} g^{3} car
73 w^{e} rb
74 w^{e} rb car
75 w^{sat}
76 w^{sat} rb
77 w^{t} fw
78 y
79 y f B
80 y g^{3}
81 y g^{4}
82 y m
83 y pn
84 y rb
85 y w m
86 y w^{e}
87 y w^{e} rb
88 y w^{w}
89 y/+

Chromosome 2

90 albasp/Cy L^{4} sp^{2}
91 a sp^{2}
92 b
93 b pr cn
94 b pr cn a
95 b pr cn vg
96 bw
97 bw^{2b}
98 bw^{4}
99 bw^{D} c
100 c
101 cn
102 cn^{35k}
103 cn vg
104 cl
105 dke c
106 lt std/Cy sp^{2}
107 lt stw^{3}
108 ltd
109 mi sp/Pm^{2}
110 pd
111 pr
112 tk sf^{2} abb
113 vg
114 vg^{dn}

Chromosome 3

115 ca
116 cd

117 cu kar
118 cu sr e^s
119 dv Mé
120 e
121 e^s cd ro cmp ca/Xa, ca
122 jv Hn^r h
123 ma fl
124 mah
125 p
126 p^p cu
127 p^p cu sr
128 p^p cu sr e^s
129 res
130 ry
131 se
132 sr
133 sr sed
134 st^{sp}
135 th st
136 th st p^p
137 th st pb p^p/Cx, D

Chromosome 4

138 ci ey^R

Multichromosomal

139 bw; ci ey^R
140 bw; e
141 bw; st
142 g^3; bw
143 g^3; st
144 g^3; st p^p
145 ras^2; st
146 rb; bw
147 rb; st
148 vg; se
149 w^e; se
150 w^e rb; se
151 y; bw; e; ci ey^R

Deficiencies

152 $Df(1)N^8$/y Hw dl-49 m^2 g^4

Inversions

153 In(1)A99b
154 In(1)dl-49, y fa^n
155 $In(1)rst^3$, y rst^3 car bb
156 $In(1)rst^3$, y rst^3 g^3 car
157 $In(1)sc^8$, w^a
158 $In(1)w^{m4}$
159 $In(1)w^{m4}$, g^3
160 $In(1)w^{m4}$; bw
161 $In(1)w^{m4}$; st
162 In(3L)pers

Translocations

163 $T(1;4)w^{m5}$
164 $T(2;3)S^M$ Cy/vg^{nw}

SPAIN

Barcelona: Centro de Genética animal y humana del Consejo Superior de Investigaciones Cientificas, Universidad

Wild Stocks

1 Oregon
2 Ujijar

Chromosome 1

3 sc ct v f car/ClB
4 w
5 y w^a cv v f

Chromosome 2

6 b cn vg
7 Cy/L^2

Chromosome 3

8 D Sb/e CL-CR
9 Gl Sb/LVM
10 ri-s se ss k e^s ro

Chromosome 4

11 ey^2

Multichromosomal

12 al L^4 Cy sp/Pm; H/Sb sr in(3R)Mé
13 cn; ss

SWEDEN

Stockholm: University of Stockholm, Institute of Genetics

Wild Stocks

1 Algeria
2 Florida
3 Karsnäs
4 Oregon
5 Stäket
6 Tunnelgatan
7 Örebro

Chromosome 1

8 B
9 Df B26-35 K/In(1)AM
10 B^i fu ♂ & y f:= ♀
11 B^n In ♂ & y f:= ♀
12 bb
14 Bx^2
15 car
16 car bb
17 cm ct^6 sn^3 ♂ & y f:= ♀
18 cv
19 cv v
20 cv v B ♂ & y.= ♀
21 cv v B car bb ♂ & y.= ♀
22 ct^6
23 ct^n
24 ct^6 v
25 ec
26 ec ct^6 g^2
27 f
28 Df f257-6/f B
29 f B
30 f B Eb/f fu
31 f B^iB^i
32 f BB/In(1)AM
33 f fu ♂ & y.= ♀
34 fa
35 g^2 B
36 g^2 f
37 g^2 f B ♂ & y.= ♀
41 lz f ♂ & y f:= ♀

42 m
43 m f
44 Df(1)N^{8}/+
45 od car
46 pn
47 rb ♂ & y f:= ♀
48 rb ct ♂ & y f:= ♀
49 sc
51 sc Sc v f car/sc^{S1} B InS
52 sc cv
53 sc cv v f
54 sc cv v f B
55 sc cv v f car
56 sc ct^{6} car ♂ & y f:= ♀
57 sc t^{2} v f Tu car ♂ & y f:= ♀
59 sc v f car
60 sc v f car/ClB
61 sc^{S1} B InS w$^{\bar{a}}$ sc^{8} (Muller 5)
62 v
64 w
65 w ct^{6}
66 w sn^{3}
67 w f
68 w^{a}
69 w^{a2}
70 w^{a3}
71 w^{a4}
72 w^{a9aV_1}
73 w^{bf} f^{5}
74 w^{bf2}
75 w^{bl}
76 w^{ch2}
77 w^{co}
78 w^{col}
79 w^{e}
81 w^{e} ct^{6}
82 w^{ec2}
83 w^{h}
84 w^{i} vb
85 w^{r} sc^{8} InS
86 w^{sat}
87 w^{t}
88 X^{c2} (closed-X)
89 y
90 y^{3P}
91 y ac sc pn ♂ & y f:= ♀
92 y ac sc pn w rb cm ct^{6} sn^{3} ras^{4} v m g f car/y sc^{S1} B InS
93 y ac sc pn w rb ct^{6} sn^{3} oc ras^{2} v m g^{2} f car/sc^{S1} B InS lz^{S}, y^{3} sc^{S1}. ♀ & y^{2} oc lz^{S}. Y^{S}/y^{3} sc^{S1}. Y^{L} ♂
94 y ac pn w rb wy^{2} g^{2}; sc^{19i}/Cy ♂ & y f:= ; sc^{19i}/Cy ♀
95 y B267-47
96 y ct^{6}
97 y f Eb/sc^{S1} B InS w^{a} sc^{8}
98 y Hw g In-49 m & y f:=
99 y rb ♂ & y f:= ♀
100 y rb ct^{6} ♂ & y f:= ♀
101 y sc
103 y sc^{5}
104 y sc^{15}
105 y sc w^{a}
106 y sc w^{e} ec rb
107 y sc^{S1} In-49 v sc^{8} ♂ & y f:= ♀
108 y sc^{S1} f In-49 m sc^{8} ♂ & y f:= ♀
109 y sc^{8} B f In-49 v ♂ & y f:= ♀
110 y $\underline{1}^{sN}$ ♂ & +.= ♀
111 y w
112 y w ct^{6}
113 y w sn^{3}
114 y w f Bx^{2}
115 y w^{a}
116 y w^{a} sn^{3} m f car
117 y w^{e} ec
118 y ec ct^{6} v f
119 y rb cm ras^{2} g^{2}; sc^{19i}/Cy ♂ & y f:= sc^{19i}/Cy ♀
120 y ct^{6} f
121 y dvr^{2} v f ♂ & y f:= ♀
122 y v g^{2} f
123 y B
124 Y^{3}

Chromosome 2

126 b
127 b j pr cn
129 b pr vg a sp
130 bw
131 dp b
132 dp pr px/Cy pr
133 L^{2}/Cy
134 pr
136 S/NS, px sp
137 sp
138 stw^{3}
139 vg

Chromosome 3

140 ca
141 D^{3}/InP
142 e^{ll}
143 e^{s}
144 gl
145 Hn^{r2}
146 Hn^{r2}; e^{s}
147 ri^{2}
148 ri^{3}
149 ro
150 ru Hn^{r2}
151 ru h st p^{p} ss e^{s}
152 ss
153 ss^{a}
154 ss e^{ll}

155 ss gl
156 st
157 st/In(3R)P
158 st ss
159 st ss e^{11}
160 se ss k e^{s} ro
161 st e^{11}
162 ve h th
163 ve h th st cu ss e^{s} ca

Chromosome 4

164 ci
165 ey^{2}
166 sv^{n}

Multichromosomal and Translocations

167 B; Cy/+
168 B; Cy/+; D^{3}/+; sv^{n}
169 B; vg
170 B; ru h st p^{p} ss e^{s}
171 +; bw^{VA}/L^{2} 1 ♂ & y f:= ; bw^{VA}/L^{2} 1 ♀
172 y v; bw^{VA}/L^{2} 1
173 b; pr; bt
174 P- T(2;3)X InCxF/Cy L^{4} sp^{2}; Dl H e p^{i}
175 +; Cy/+; D^{3}/+; sv^{n} ♂ & +.=; Cy/+; D^{3}/+; sv^{n} ♀
176 B; sv^{n} ♂ & +.=; sv^{n} ♀
177 pr; ss
178 bw; st
179 L^{2} sp; th
180 sc cv v; ri^{3}
181 sp; th
182 T(1;2)7/ClB
183 T(1;2)B^{bd7}/Cy ♀ & M2e/Cy ♂
184 T(1;2)Bld/Cy
185 T(2;3)bw^{VDe4}/Cy
186 w^{co}; D^{3}/+
187 y; pr; ss
188 y; ro
189 y^{Si} sc^{8} InS y^{3P}; al^{2} Cy lt^{3} sp^{2}/dp b Pm^{1}; ru h D InCxF ca/Sb In 3R
190 y ct^{6}; ro
191 y; ca

SWITZERLAND

Basel: Botanische Anstalt der Universität

Wild Stocks

1 Basle
2 Oregon-R-C
3 Tessin

Chromosome 1

4 B
5 ClB/lz w
6 f
7 v
8 w^{a}
9 w^{co}
10 w^{co} sn^{2}
11 w^{e}
12 y w^{a} cv v f

Chromosome 2

13 b vg bw
14 Cy/Bl sp^{2}

Chromosome 3

15 DcxF/Dfd
16 se cp e

Multichromosomal

17 Cy/Pm^{d33k}, H/Sb C
18 y Hw m^{2} g^{4}, dl-49

Unanalyzed

19-39 Different mutant stocks obtained by X-raying

Zürich: Zoologisches Institut der Universität

Note: No new stock list received. See DIS-22, p. 51.

NEW MUTANTS

Report of Arvelighetsinstituttet, University, Oslo

dp^{51f}: dumpy^{51f26} Mossige. 2-13.0. Spontaneous in y stock. Extreme dumpy wings and vortices. Viability and fertility good. RK1.

j^{49j}: jaunty^{49j18} Mossige. 2-48.7. Spontaneous as 1 female in sv^{2} stock. Less extreme than jaunty. Some overlapping at 21°, none at 30°. Viability and fertility good. RK2.

j^{50e}: jaunty^{50e5} Mossige. 2-48.7. Spontaneous in sc cv v f stock. Like j^{49j}. RK2.

lz^{51d}: lozenge^{51d10} Mossige. 1-27.7. Spontaneous as single male in st stock. White eye in combination with st. Eye extremely reduced in size, smooth and glossy. Color light yellow with brownish rim and spots. Female sterile. RK1.

M(2)50j: Minute(2)50j7 Mossige. 2-55±. Spontaneous in Kiil's "ru notch" stock. Medium bristle effect. Reduces crossover distance between pr and cn to 1.5. Not deficiency for rl but lethal with M(2)S10. RK1.

ma^{49d}: maroon^{49d} Oftedal. 3-49.7. Spontaneous in Hn^{r} h stock. Possibly identical with jelly (lost) which appeared in same stock before the war. Eye color fairly like bw in young flies, darkening to se in very old flies. After isolation showed several pheno-variations, none of which could be established. RK1.

ms: missing^{50b4} Mossige. 2-(not located). Spontaneous in na/sc^{8} dl-49 B w^{a} lx^{s} stock. Bristles very reduced or missing, in extreme cases almost like sv. Females sterile, males low fertility.

tu(2)49k: tumor(2)49k Oftedal. 2-(probably between c and px). See Research Notes. Appeared spontaneously as black aggregations in abdomen or thorax in flies of ma^{49d} stock. Tumors visible in larvae 96 hours after hatching. Incidence variable, approaching 100% in uncrowded cultures. Incidence during one 4-month period 56%, somewhat higher in females than in males. Modifiers located in Chromosome 1 and middle of Chromosome 3.

w^{50j4}: white^{50j4} Mossige. Spontaneous in M(2)l^{2}/Cy, L^{4} stock. Like w. RK1.

Report of Instituto de Genética, University of Buenos Aires

b^{50b}: black^{50b} Fernández Gianotti. 2-48.5. U. Spontaneous in San Miguel (Prov. of Buenos Aires) wild stock. Like b. RK1.

f^{50c}: forked^{50c} Fernández Gianotti. 1-56.7. U. Spontaneous in Tunuyan (Prov. of Mendoza) wild stock. Like f. RK1.

od^{50a}: outstretched^{50a} Fernández Gianotti. 1-59.2. U. Spontaneous in San Miguel (Prov. of Buenos Aires) wild stock. Like od. RK2. Lost.

se^{50b}: sepia^{50b} Fernández Gianotti. 3-26.0. U. Spontaneous in Cinco Saltos (Rio Negro) wild stock. Like se. RK1.

y^{50a}: yellow^{50a} Fernández Gianotti. 1-0.0. U. Spontaneous in San Miguel (Prov. of Buenos Aires) wild stock. Body and wings yellow color, hairs and bristles dark color. RK1.

y^{50b}: yellow^{50b} Fernández Gianotti. 1-0.0. U. Spontaneous in Tunuyan (Prov. of Mendoza) wild stock. Like y. RK1.

y^{50b25}: yellow^{50b25} Fernández Gianotti. 1-0.0. U. Spontaneous in Tunuyan wild stock. Body gray, wings yellow, and hairs and bristles dark. Lost.

Report of Istituto di Genetica, University of Milan

mwh: multiple wing-hairs Di Pasquale, 501. Spontaneous from the wild stock Moltrasio. Hairs on the wings grouped in number of 2-5 in the place of I. Recessive, chromosome 3, locus not determined. Viability excellent and penetrance complete. RK1.

Report of M. M. Green

su^{3016}-v: suppressor of vermilion. 1-0.0. X-ray. Arose as single male from X-rayed sn^3 lz^{BS} v ♂ crossed to XX: y f ♀♀. Allelic to su^2-s but not tested for suppression of sable. RK5.

Report of P. T. Ives

Cy bw^{45a} sp^2 or^{45a} Ives. Reported in DIS-19, p. 46, as Cy bw sp^2. This chromosome has since been shown to carry or as well as bw, but it does not carry pd. Cy bw^{45a} sp^2 or^{45a}/or = or; Cy bw^{45a} sp^2 or^{45a}/bw = bw; bw^{47j}/or = +. Since, in addition, Amherst's Cy sp^2, Cy L^4 and Cy Bl L^4 chromosomes have been shwon not to carry or (Mossige, DIS-24, p. 61), the suggestion is that bw^{45a} and or^{45a} represent a double mutation in the Cy sp^2 chromosome. Neither is lethal when homozygous, but they have not been crossed away from In(2R)Cy. Since cn^2 is also present, the homozygote is yellow-to-orange (with sp^2 wings). The left arm of this chromosome carries at least one lethal gene near each end of In(2L)Cy: l^{1076} and l^{1323}, respectively. It is proposed that this chromosome, for simplicity, be designated Cy bw sp^2 or. It has excellent viability as Bl/Cy bw sp^2 or.

l^{1076}: lethal^{1076} Ives, 49h. Extracted from the South Amherst wild population in form Bl/l. Cy bw sp^2 or/l^{1076} is lethal. l^{1076} is about 40 units from Bl. Since 2R of Cy bw sp^2 or is not lethal when homozygous, this indicates l^{1076} is allelic to a lethal which is in Cy bw sp^2 or, near the left end of In(2L)Cy.

l^{1323}: lethal^{1323} Ives, 51g. Extracted from the South Amherst wild population, in form Bl/l. Cy bw sp^2 or/l^{1323} is lethal. l^{1323} proved inversion-free when tested for crossing over with net b cn bw, but showed no crossing over against Bl in 162 test flies. Thus this lethal is allelic to one which is in Cy bw sp^2 or, near the right end of In(2L)Cy.

or^{49h}: orange^{49h31} Ives. Extracted from the local wild population. A bw-like color, the same phenotype as or; or^{49h}/or = or. Also, or^{49h}/Cy bw^{45a} sp^2 or^{45a} = or. RK1.

Report of George Lefevre, Jr.

Four duplications of the white locus, inserted into autosomes, have been produced by irradiation. These are carried as males in the University of Utah stocks with a white-deficiency rst^3 X chromosome mated to y w f:= females. The first has been reported previously (DIS-23, p. 59), but is now symbolized:

Dp(1;3)49a7 (Spotted) Lefevre. A duplication of the w-spl region inserted into the heterochromatin of 3; shows mottling for w, rst, and spl. Shorter than Dp(1;3)N264-58a of Demerec, which extends from 3B3-4 to 3D5-6 inclusive; but exact extent undetermined. Although mottled for the visible loci, covers many white and Notch lethals, as well as rst^2, but did not cover the X-deficiency (now lost) from which it was derived. Induced in Canton-S.

Dp(1;f?)50k11 Lefevre. A non-mottled duplication of the w-rst region. Appears to assort independently of bw, e, and ey. Does not cover pn or spl. Poor viability in normal males, but fairly good with w deficiencies. Was found in a Notch fly, and does not cover the original deficiency from which it was derived. Induced in Canton-S.

Dp(1;?)51b7 Lefevre. A non-mottled duplication of the w-spl region, produced by irradiation of w^{m4}. Position not yet determined. Effectively covers white and Notch lethals, but not the deficiency from which it was derived.

Dp(1;?)51c20 Lefevre. A mottled duplication of the w-rst region, produced by irradiation of w^{m4}. Position not yet determined. Covers some white lethals, but not the deficiency from which it was derived.

Report of Helen U. Meyer and Margaret Edmondson

b^{50d}: black^{50d} Meyer, 50d. 2-48.5. Ultraviolet-induced, allele of black, somewhat lighter phenotype. Present in b^{50d} crs/Th Cy Bl L^4 sp^2 stock. RK2.

b^{51f}: black^{51f} Meyer, 51f. Ultraviolet-induced. Allele of b and phenotypically like b^{50d}. Also present in combination with crs. RK2.

che: cherub Meyer, 48g. 2-62.± 1. Ultraviolet-induced, wings short, papery, and downcurved, with short broad alulae. Localization of the mutant showed that the same chromosome contained a semilethal nearby, near 60, and also a male- and a female-sterility mutation, at different loci from each other. The male-sterility locus is probably the same as that of the mutant; that is, male sterility is probably an expression of the mutant gene (see description of che^2). Homozygotes for che are short-lived, and even the balanced stock cn che bo sp/al^2 Cy cn^2 L^4 sp^2 requires 30% longer than normal for a generation.

che^2: cherub^2 Meyer and Edmondson, 48l. Ultraviolet-induced. An allele of cherub, with similar wing characters and male sterility. Also induced in the same chromosome is a mutant which causes spread wing, located to the left of che^2, near 60. Homozygous females fertile. Since it has been shown by Edmondson that the male sterility here is also an allele of that in che, the wing character and the male sterility are probably expressions of the same mutant gene (unless due to a very minute deficiency). Present in cn che^2 bw sp/al^2 Cy Bl L^4 sp^2 stock, with normal heterozygous viability and generation time. RK2.

dp^{o51e}: dumpy-oblique^{51e} Byers and Meyer, 51e. Ultraviolet-induced or spontaneous. Found in all the offspring from a given male (treated in polar cap stage) which also carried a lethal in the same second chromosome. Allele of dp, showing comparatively slight wing effect, but not vortex effect, in compound with dumpy and with truncate, and normality in compound with thoraxate, all three compounds being viable. Present in dp^{o51e} ab^2 cn sp/ Th Cy Bl cn^2 L^4 sp^2 stock.

dp^{Th51e}: dumpy-thoraxate^{51e} Meyer, 51e. Ultraviolet-induced or spontaneous, more likely to be induced (although present in all functional germ cells left after UV treatment of the pole cells), since a lethal was similarly present in the homologous second chromosome in all gametes which received that chromosome. Lethal as homozygote, and in compound with dp^{Th}. dp/ dp^{Th51e} is viable, shows vortices of bristles and hairs on thorax and some blistering of wings, but wing shape not affected. Present in stock dp^{Th51e} ab^2 cn sp/al^2 Cy Bl L^4 sp^2; RK1 as a lethal.

ds^{51a}: dachsous^{51a} Meyer and Byers, 51a. 2-0.3. Ultraviolet-induced; found to be allele of ds^{33k}, phenotype resembles description of ds^d; sex combs were observed to be missing in 2 male mutants. Owing to a pupal near-lethal at locus 25.5, viability was very much reduced; homozygotes (including also this near-lethal) required longer time for development and were short-lived. RK4.

fs 2.1: female sterile in second chromosome Edmondson, 50j. 2-57.6. Ultraviolet-induced. Completely sterile females; lay no eggs; gonads rudimentary (examination by Meyer). Induced in the same chromosome was a male-sterility gene, located between L and a. Localization of fs 2.1 based on 60 crossovers between Bl and L. Present in fs 2.1 sp/Cy Bl cn^2 L^4 sp^2, bur fs 2.1/Cy cn^2 L^4 sp^2, and fs 2.1 bw sp/Cy Bl cn^2 stocks. RK1.

fu^{51e}: fused^{51e} Edmondson, 51e. Ultraviolet-induced. An allele of fu, and like the published description of the original fused, with marked fusion of L_3 and L_4 veins. Wings much extended, ocelli reduced, and bristles of ocellar region mostly lacking. Eyes rather small and roughish. fu^{51e} males are fertile, fu^{51e}/fu^{51e} females sterile with fu^{51e} males. Our present stock of fu, f fu/ClB, is not so marked in any of its characters, and the f fu/fu^{51e} compound is like the f fu parent, the wings being only slightly spread and there being only a slight loss of ocellar bristles and ocelli. The fusion of the L_3 and L_4 in the compound still eliminates the anterior crossvein, but thereafter L_3 and L_4 are separate until very close to the wing margin. Present in fu^{51e}/y sc^{S1} B In49 v stock. RK1.

l(2)gl: lethal (2) giant larvae^2 Meyer, 51a. Ultraviolet-induced. Phenotype of larvae like that described by Bridges for l(2)gl; larvae fail to pupate and become bloated to giant size, die eventually. Assumed to be allelic; locus given by Bridges as 2-0±, which seems to agree with our crossover counts. Localization showed that besides the giant-larvae lethal (recognized because the balancing chromosome contained an allele; see $l(2)gl^3$ the same chromosome had an embryo lethal not separated from the Star locus. The latter roughens the surface of the eye in heterozygotes, but although itself lethal when homozygous is not lethal in heterozygotes having Star from other parent. Also present, located in the region near cn, is a mutation which makes the homozygous flies die not later than 2 days after hatching. A dominant suppressor of Sp is also present and a mild allele of dumpy, probably dp^{o4}, which causes the wing of dp^{o4} ta homozygotes to appear extremely narrow. There is also a female-sterility mutant at the left end of the chromosome, which is expressed by the reduction in size of the ovaries; and, though germ cells are present, there are only one or two eggs having the usual bulk of nutrient material. Present in ta cn bw/Cy cn^2 stock with the above mentioned mutants in the ta cn bw chromosome, an extremely weak stock.

$l(2)gl^3$: lethal (2) giant $larvae^3$ Meyer, 51a. Spontaneously in Cy cn^2(ins R and L)sp balancer. In combination with the UV-induced $l(2)gl^2$ acts exactly like Bridges' original lethal, present in H. J. Muller's stock i 47. RK1 as lethal.

lt^5: $light^5$ Meyer, 51d. 2-55.0. Ultraviolet-induced; allele of lt^3 and lt^4, and, like the latter, gives lethal effect when homozygous. Compound lt^3/lt^5 viable with mutant eye color; lt^4/lt^5 lethal. The fact that both lt^4 and lt^5, induced by UV in experiments done a year apart, give allelic lethal effects and are at the same time alleles of the visible mutation lt^3, and that in the case of lt^4 the lethal was found to be so close to the lt locus that it had not separated in crossover tests (DIS-24), makes it likely that the lethal effects of both are due either to a gene mutation at the lt locus (see Muller and Valencia's parallel cases of lethal raspberries and carnations) or to deletions which include the lt locus. Present in stock lt^5 crs/Th Cy cn^2 Bl L^4 sp^2. RK1 as lethal.

N^{51d}: $Notch^{51d}$ Byers, 51d. 1-(not localized). Ultraviolet-induced. Present in N^{51d}/y sc^{S1} B In49 v stock. RK1.

sps: spastic Edmondson and Meyer, 49d. 2-63.6. Ultraviolet-induced. Pupal and post-pupal lethal. Many flies emerge from pupae and wings expand normally, but flies are unable to walk or fly, owing to spastic contraction and jerking of leg and wing muscles. The spasticity in the legs results in flies' flipping over on their backs and becoming stuck in that position. In any event they die within 24 hours, and do not breed. Under ether the muscles relax so that flies become indistinguishable from normals. A lethal, at locus about 6.3, was also induced in this same chromosome. Present in isogenic cn sps bw sp/Cy cn^2 L^4 sp^2 stock. Rk1 as lethal.

stw^5: $straw^5$ Meyer, 51d. 2-55.1. Ultraviolet-induced. Allele of stw^3, semilethal or associated with a semilethal close by. In stw^5 ta cn bw sp/Th Cy Bl L^4 sp^2 stock. RK3.

stw^6: $straw^6$ Meyer, 51e. Ultraviolet-induced in a crs-bearing chromosome. Allele of stw, viability better than stw^5, but also reduced. RK2.

tom: tomboy Meyer, 50k. 2-61.5±2. Ultraviolet-induced. Females homozygous for tomboy have male-like pigmentation of the posterior tergites and often a lump of extra tissue with or without claw-like formations in the genital region. Best expression at 27° C, but almost lethal, whereas at 17° C the expression is reduced and the viability improved. These females have rudimentary ovaries and are sterile. Male homozygotes phenotypically unchanged (sex combs present) but have non-motile sperm. This mutation was induced in a chromosome carrying the gene for $abrupt^2$. ab^2 when homozygous makes males near-sterile by causing rotation of the genital region, although functional sperm are present. The sterility gene which is with the tom mutation then adds another type of sterility. The possibility is not excluded that this new male sterility may be at another locus, but it seems likely that the female sterility, at least, might be due to the action of "tomboy" itself. In females homozygous for ab^2 and tom the tendency of the ab^2 gene to rotate the genital region (of males) here manifests itself in rotating the lump of extra tissue with its claw-rudiments caused by the action of the tom gene. Viability between 10% and 30% of expectation, depending on temperature. Present in stock ab^{2} tom bw sp/Th Cy Bl L^{4} sp^{2}. Rk2 as mutant.

v^{51g}: vermilion^{51g} Edmondson, 51g. Ultraviolet-induced. Phenotypically similar to and allelic with v. Also induced in the same chromosome was a pupal lethal. Present in v^{51g}/y sc^{S1} B In49 v stock. RK1.

Report of George H. Mickey

ct^{50e}: cut^{50e} Bakkum, 50e. 1-? Extracted from LaCrosse wild-type stock. Wings cut to points; eye slightly ovoid. Viability and fertility lowered, especially in females. Salivaries not analyzed. RK2.

di^{49b}: distorted eye^{49b} Mickey, 49b5. 1-23 approx. Single male in F_2 from female treated as second-instar larva with natural gas for 6 hours. Eye reduced and roughened. Wide variability of expression--whole or part of eye being roughened, usually lower half but occasionally a vertical bar. Also sometimes affects bristles (scutellar, postvertical, dorsocentral, and supraalar either absent or doubled), ocelli (some missing), and wings (rough surfaces or nicked margins and plexus veins). Expressivity variable, showing better in progeny of young females, but not altered by temperature or culture conditions. Viability and fertility high. Crossover tests by Don Ede show erratic results, with tendency for increased values in regions adjacent to di. Salivary chromosomes appear to be normal. RK5.

N^{50b}: Notch^{50b} Mickey, 50b. 1-1.5 to 1.7. Spontaneous in y stock. Typical Notch phenotype. Deficient for w and rst. RK1L.

R^{51b}: Roughened^{51b} Mickey, 51b21. 3-1.4. Pub. 1951, Rec. Gen. Soc. Amer. 20: 113-114. Arose as a mosaic male from a female treated as 2-hour egg with cold shock (-4° C) for 1 hour. Heterozygote shows irregular arrangement of facets and hairs on eyes, but facets not fused. Eye reduced and oblong, with spectacle effect along ventral border; color wild type; ocelli unaffected. Viability normal. Homozygous lethal. R/R^{51b} shows greatly reduced eye and more extreme facet alteration; much fusion, resembling gl and Gl. RK1.

w^{51f}: white^{51f} Di Paolo, 51f. 1-? Spontaneous in Muller-5 stock. Unanalyzed. RK2.

Report of Sidney Mittler

tu^{50j}: benign melanotic tumor^{50j} Mittler, 50j20. Found upon inbreeding wild Chicago '50. Excellent fertility and viability. Penetrance 5-10% when S. cerevisiae is used as yeast on cornmeal-molasses. Black pigment spots occur usually on ventral side of abdomen, equally in both sexes.

Report of E. Ohnishi

b^{51i}: black^{51i18} 2-48.5. In N-X stock. RK1.

e^{51g}: ebony^{51g} 3-70.7. In fw^{50k} stock; as black as e^{11}. RK1.

fw^{50k}: furrowed^{50k16} Allelism not tested, but typical fw. RK1.

H^{51c}: Hairless^{51c2} In cross Senzoku wild x Bd/+. RK1.

v^{50k}: white^{50k16} 1-1.5. In N-X stock. RK1.

v^{51a}: vermillion^{51a10} 1-33.0. In Bd/+ stock. RK1.

v^{51b}: vermillion^{51b25} 1-33.0. In vg^{ni} stock. RK1.

Report of S. Rosin

1(2)46i: lethal (2) 46i Rosin, 46i. 2-57.0±0.6. Isolated from 1(3)tr/Mé Sb. RK1.

drb: dark red-brown Rosin, 48b. 3-47.7±. Spontaneous in wild stock. At 18° C eye color dark red-brown, at 28° C dark red. drb/+ at 18° C darker than wild, but at 28° C not more distinguishable. Crossing over between st and p reduced to 50% and between p and ss reduced to 70% of normal. RK2A.

Pl: Pearl Rosin, 1948. 2-6±. (Crossing over data with S not yet homogeneous). Pub. and fig. 1951, Rev. Suisse Zool. 58: 398-403. Spontaneous. Pearl-like nodes in the wings. Wing margins often snipped and venation disturbed. Defects in bristle pattern. Eyes small and rough. At 28° C, at least one of these characters always present. At 18° C, almost completely normal. Viability good, fertility of males perhaps somewhat reduced. Expressivity dependent upon modifier genes (Nef). Cytological: partial cell decay in all imaginal discs. In the wing disc, vital cell complexes are isolated by dead cell masses. They organize themselves to inverse epitheleal spheres which furnish the pearls. Homozygous lethal at early pupal stage (Tschanz). RK3 at 28° C.

Report of H. Slatis

bw^{59}: brown^{59} Slatis, 50f16. From X-rayed st male crossed to bw; st female. With st the homozygote is browner than +. bw^{59}/bw; st has dark brown pigment. RK1.

bw^{75}: brown^{75} Slatis, 50f25. From X-rayed st male crossed to bw; st female. With st the homozygote is brownish purple. bw^{75}/bw; st is tan. Twin spots of dark color and white in the eye of a few bw^{75}/bw; st flies suggest that this allele can be used to show spontaneous somatic crossing over. RK1.

bw^{81}: brown^{81} Slatis, 50h1. From X-rayed st male crossed to bw; st female. With st the homozygote is orange. bw^{81}/bw; st is very light orange. RK1.

These three stocks do not form a single series intermediate between wild-type and the recessive, but appear to be representative of more than one such series.

Report of F. H. Sobels, Elze C. Botcrenbrood, J. Faber, and F. J. Oppenoorth

Asy: Asymmetric Sobels, 49j. Continued analysis on the phaen abnormal tergites necessitate a correction of the report in DIS-24. Replacement experiments with dominant marker genes for the different chromosomes, carried out on extensive material, did not allow a positive conclusion as to the chromosomal localization of the responsible factor. This result, as well as the increasing frequency after numerous generations of selection and the low frequency in the F$_2$ and F$_3$ offspring of inbreeding after outbreeding to a wild-type Berlin Inzucht stock, all indicate that these abnormalities are determined by polygenic factors. Components of this polygenic complex show dominance. A specific lethality, which could not be ascribed to unfertilized eggs, was observed. In all crosses the favoring effect of Dichaete on manifestation frequency was evident. Presence of a sex-linked modifying gene could not be established; penetrance, however, is better in males (±16%) than in females (±9.5%). Manifestation frequency of head and thorax abnormalities was somewhat decreased in the stock D/Mé Sb inbred for tergite abnormalities during more than 40 generations, but still remained present. Hence, our conclusion that dorsomedian fusion of the hypoderm and proper evagination of the dorsal imaginal discs are governed by a connected polygenic system, seems to be correct.

dp^{50c}: dumpy^{50c} Sobels, 50c7. Appeared in a ltr/Mé Sb stock. Manifests only with Mé present. Homozygous viable; slight dominance, which is significantly higher at 28°. Pm also acts as intensifier. In homozygous condition best penetrance at 25°, varying from 54% to 98%, also higher than at 28°; no manifestation at 16°. Manifestation is better in females than in males. Expressivity also greatly variable. A significant positive correlation exists between penetrance and expressivity. Exact localization proved to be difficult on account of varying penetrance and absolute dependence on presence of Mé. Tests of allelism rendered no useful results because of the dominance of dp in the presence of Mé. Striking resemblance to dp^{To} and other dp alleles, however, makes location at dp locus highly probable. RK4.

l(1)ne: lethal non-evaginated Vogt, 1949. 1-0.1±. Appeared as a urethane-induced mutant in M. Vogt's experiments and was localized and studied in the Utrecht laboratory. Lethality occurs in the late pupal stage. The puparium is more darkened than normally and preserves its prepupal shape. The tracheal trunks are meandering, which forms quite a safe character for distinguishing lne prepupae from normal ones. The head and thoracic imaginal discs show almost complete imaginal differentiation, but evagination fails to occur. This may perhaps be due to a premature disintegration of the larval hypoderm in the thoracic region, which is observed about 4 hours after pupation. Because of the perfect differentiation of the abdominal hypoderm the animals are characterized by a sharp, darkly chitinized, duplicated border between the thorax lacking its integument and the normal abdomen. The genital disc also metamorphoses, the hypopygium often shows torsion; ducti efferentes are mostly underdeveloped. The testes do not get connected with the ducti efferentes and, in consequence, never spiralize. The brain metamorphoses; the imaginal differentiation of the ventral ganglion, however, is blocked at a prepupal stage. The alimentary canal is almost completely differentiated. After transplantation into normal hosts, eye, wing, haltere, and leg discs also show perfect imaginal differentiation. Ring-gland implantation in lne larvae causes no alteration in external appearance of the pupa. RK1.

Report of Bentley Glass

ap^{49j}: apterous^{49j} Ritterhoff, 49j. 2-55.4±. Spontaneous in car

$l^{(B5+2)}$/Ins(1)sc^{S1} B sc^{8} w^{a} stock. Wings and halteres reduced to vestiges. Bristles and microchaetes on sides absent, including p. notopleurals, a. and p. supra-alars, and a. postalars; but dorsocentrals are not reduced in size and number, and one or a pair of pre-anterior dorsocentrals may be present; p scutellars are not absent nor erect when present, but normal; hairs on back of thorax are normal. Sutural furrow normal, thorax not reduced in size. Viability rather better than previous ap alleles described, but both sexes still sterile. Not tested with ap or blt for allelism, but heterozygote with Xa has bloated wings, about those of Xa in size, which burst, turn black, and shrivel to vestiges. This test further indicates that ap^{49j} is a less extreme allele than ap^{4}.

lz^{50d}: $lozenge^{50d}$ Ritterhoff, 50d. 1-27.7. From X-rayed f B male. Like lz^{s} in phenotype and female-sterility.

vnl: venula Plaine, 50h. 2-?. Spontaneous in b $Su\text{-}er^{+}$ bw; st er stock. Wings with extra veinlets located between L_3 and L_4, largely between ant. and post. crossveins; additional veinlets frequently arising from L_4 distal to post. crossvein. Wing size (length and width) slightly reduced. Not allele of dsr. Penetrance in male, .013; in female, .43. With Sb, penetrance in female increases to .63; probable En-vnl in chromosome 3. Expressivity also enhanced.

Report of A. J. Bateman

Additions and Corrections to Report in DIS-24

aw^{1} Linkage test repeated at lower temperature of development (so as to give 70% penetrance). This gave a value of 32. Not allelic to aw^{2} (q.v.). Viability 47% under optimal conditions. The nearest previously recorded mutant of similar phenotype, dvr (divers), at 28.1 is not available for test of allelism. But whereas y produces marked increase in expressivity of dvr it has no effect on aw^{1}.

aw^{2} Not allelic to aw^{1}, though phenotypically similar. Penetrance nearly complete at 25° C, and viability 10% under optimal conditions, with failure of most males to emerge from pupae. Located more accurately at 38-39. Does not appear to correspond to any previously described mutant.

Bt Observations on salivaries by Mr. L. A. Darby established that there was a duplication in tandem (not an inversion) of the moderately large segment 3C1-6F6, which paired with itself, forming a loop of triple thickness. The phenotype is in fact similar to some hyperploids.

fd Salivary studies by Mr. L. A. Darby showed X-3 interchange, with the breaks at 7A4 in X and 86E8 in 3. Besides this there was an inversion in 3 near to the point of interchange, 89C6-96A20. This inversion, together with the high proportion of nondisjunction, leads to low c-o values in the X, so it has been impossible to locate the fd mutant accurately (assuming fd to be a point mutation). It is probably at or near the interchange.

N^{P} Salivary studies by Mr. L. A. Darby showed an inversion from 3C (at or near fa locus) to 8E.

rg^{P}: $rugose^{P}$ (Originally fas: facetious.) Viability 75% under optimal conditions. Salivary studies by Mr. L. A. Darby revealed a small inversion 3C-4E. Since 4E is near the locus of rugose, it was tested and found allelic to this mutant, which is probably at the point of the right break.

New Mutants

rb^{50j}: ruby^{50j} Arose in son of a Samarkand control male by $\overline{XX}$ female. Typical allele. Viability, penetrance, and fertility 100%. RK1.

un^{P}: uneven^{P} Arose in son of P32-treated male by $\overline{XX}$ female. Phenotype and linkage indicate "uneven." Viability, penetrance, and fertility 100%. RK1.

v^{P}: vermilion^{P} Arose as two sons of P32-treated male by $\overline{XX}$ female. Typical allele. Discarded.

g^{P2}: garnet^{P2} Arose in son of P32-treated male by $\overline{XX}$ female. Typical allele. Discarded.

crm: cramped 1.1±. A semilethal from P32-treated male through Muller-5. Antennae are stumpy with shrunken warped aristae, usually lying back on head. When it first appeared the wings were narrow and scalloped and the eyes were pear shaped, with concave sides and top of pear pointing downwards. Wing effect rapidly disappeared as viability rose to 15%. Antennal effect constant. Sterile. Gave no c-o with w in 254 males. RK3.

vs^{P4}: vesiculate^{P4} Reoccurrence of vs from P32-treated male through Muller-5. Low expressivity and penetrance, and viability 60% under optimal conditions. Crossing over normal from vs to right end of X but very low from sc - vs. Mr. L. A. Darby found from salivaries a segmental interchange involving left end of X (to vs locus?) and most of 2L. A position effect? RK4A.

lz^{50j}: lozenge^{50j} Arose in same chromosome as a lethal (at 6.0) from an X-rayed male through Muller-5. During linkage tests some extreme lz flies appeared as crossovers. Males fertile. Females sterile, dying at maturity. RK1.

In addition to the above, the following mutants reverted to wild-type too quickly for naming to be justified.

(a) Rough-eyed autosomal dominant from P32-treated male by $\overline{XX}$ female. At first penetrance and expressivity excellent. Located on chromosome 2. Then penetrance rapidly deteriorated. Discarded.

(b) Autosomal dominant from X-rayed male by $\overline{XX}$ female. L5 shortened, sometimes L4 also. Eyes sometimes rough and misshapen. Penetrance higher in females. Located in left end of chromosome 2. Discarded.

(c) Sex-linked recessive from P32-treated male extracted with Muller-5. Arose in same chromosome as a lethal from which it was separated by c-o during a linkage test. It appeared first in combination with v f car. Penetrance and expressivity were then extreme. Eyes small and rough; wings thin, outstretched, and often curled downwards, with edges corroded; genital arches reduced to vanishing; many thoracic bristles reduced or missing (especially notopleurals, supra-alars, dorsocentrals and scutellars). Viability 10%, short-lived, sterile. Located at 24 ±. During linkage test mutant flies separated from v f car approached wild-type phenotype, and were more viable and sometimes fertile. The only remaining reliable characteristic was the irregular reduction or removal of thoracic bristles. Resembles reduced (rd) in chromosome 2

LINKAGE DATA

Report of R. Milani. The locus so of Drosophila melanogaster.

The gene so (sine oculis, Milani, DIS-14) has been localized, using the two alleles so and so^{1}. Most of the experiments have been done using the mutants in coupling with the markers; however so has also been checked in repulsion. In this case the testcross was: + so + +/b + cn vg x so^{1} cn vg bw/ so^{1} cn vg bw. This cross allows easy detection of the so cn chromatids because the so/so^{1} heterozygotes have eyes (often deformed). The localization is based on the scoring of a gross total of 6185 flies. The markers used were b, cn, vg. The order is b, so, cn, vg; and the results can be summarized as follows:

Markers	Locus	Crossover rate of so	Locus of so	$\frac{p\cdot(1-p)}{n}$
b	48.5	8.9	57.4	± 0.4
cn	57.5	0.4	57.1	± 0.1
vg	67.0	10.8	56.2	± 0.4

Considering the nearer marker, the gene results located at a distance of 0.4 from cn, on the side of the centromeres; its locus should therefore be at point 57.1 ± 0.1. The crossover rates of the other markers are in good agreement with this hypothesis.

Report of E. Ohnishi. Location of Notch-Xasta (N-X, reported in DIS-24).

First experiment which crossed to se ss ro was unsuccessful by the presence of an inversion in 3R. But a few (6/1446) showed crossing over and these were crossed to Canton-S and backcrossed by se ss ro.

Since recombination values were N-X ro, 38.1%; N-X ss, 7.6%; ss ro, 32.2%; the locus of N-X is 3-50.9. Penetrance is to be changed to 100% (50%, DIS-24). Homozygous semilethal and phenotype of survivor is like Xasta of Serbrovsky. Viability of heterozygote is fairly good. Expression of heterozygous N-X is enhanced by Dl and supressed by H. Combination of N-X and Xa resulted in considerable decrease in viability and tiny vg-like wing. Good as dominant marker. RK1 (instead of RK3 as reported in DIS-24).

N-X ss ro/	x ss ro
N-X ss ro	548
	659
ro	333
N-X ss	294
ss ro	68
N-X	60
ss	15
N-X ro	8
	1985

STOCK LISTS

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

D. pseudoobscura

Wild strains homozygous for different gene arrangements in the third chromosome

Piñon Flat, California:
- Standard-14 strains
- Chiricahua-11 strains
- Arrowhead-3 strains

Mutants

Chromosome 1

y sn v co sh

Chromosome 2

up bx Ba gl inversion/lethal

Chromosome 3

or Bl Sc pr/lethal Cuernavaca

Chromosome 4

in hk j Cy/in hk j
in hk j Cy inversion/+

Multichromosomal

Ba; or; Cy

BALTIMORE, MARYLAND: THE JOHNS HOPKINS UNIVERSITY

D. funebris
D. simulans
D. simulans: Lima Peru a^5
D. simulans: Lima Peru a^{10}
D. simulans: New Orleans-51
D. simulans: Los Angeles-50
D. virilis

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA

D. simulans
D. virilis: Pasadena 10^k
D. virilis: Pasadena wild
D. virilis: scarlet

BLACKSBURG, VIRGINIA: VIRGINIA POLYTECHNIC INSTITUTE

D. robusta

Wild Strains

N.Y. City area, 1947-49:
- 6 strains homozygous for arrangement 2L
- 8 strains homozygous for arrangement 2L-1
- 5 strains homozygous for arrangement 2L-3
- 9 other strains

Blacksburg area, 1950-51:
- 43 strains

CAMBRIDGE, MASSACHUSETTS: HARVARD UNIVERSITY

D. persimilis

Wild Strains (California)

16 strains (Jacksonville)
32 strains (Mather)
20 strains (Mono Lake)
18 strains (Timberline)

Also 6 unstudied autosomal recessives.

Chromosome 2

up bx Ba gl/inversion

Chromosome 3

or

Chromosome 4

Cy

EVANSTON, ILLINOIS: NORTHWESTERN UNIVERSITY

D. affinis (Wilmette, Ill., 1950)
D. buskii (Evanston, Ill., 1949)
D. hydei (Wilmette, Ill., 1950)
D. immigrans (Wilmette, Ill., 1949)
D. simulans (Evanston, Ill., 1949)

KNOXVILLE, TENNESSEE: UNIVERSITY OF TENNESSEE

D. funebris

Wild Stock: Knoxville, Tennessee (1948), single female strain

D. hydei

Wild Stocks: Single female strains taken at various altitudes in Great Smoky Mountains, Tennessee (1950-51)

D. immigrans

Wild Stocks: Single female strains taken at various altitudes in Great Smoky Mountains, Tennessee (1950-51)

D. robusta

Wild Stocks: Single female strains taken at various altitudes in Great Smoky Mountains, Tennessee (1950-51)

D. simulans

Wild Stock: Knoxville, Tennessee (1949), single female strain

LINCOLN, NEBRASKA: THE UNIVERSITY OF NEBRASKA, ZOOLOGY DEPARTMENT

D. affinis: Nebraska (1)
D. algonquin: Nebraska (1)
D. athabasca: New York (2)
D. athabasca: New Jersey (1)
D. melanica: Nebraska (2)
D. robusta: Nebraska (3)

Mutant strain: reduced eyes (re)

LOS ANGELES, CALIFORNIA: UNIVERSITY OF CALIFORNIA, BOTANY DEPARTMENT

D. repleta (group)
D. busckii
D. immigrans

D. pseudoobscura

Wild Strains. Structurally homozygous third-chromosome gene arrangements, derived from wild females collected at five stations equidistant along a fifteen-mile transect in the San Jacinto Mts., Southern California. New strains are being added. Stocks from Mather, California, are also maintained.

Standard gene arrangement	Chiricahua gene arrangement
Keen Camp-16 strains	Keen Camp-12 strains
Thomas Mt.-14 strains	Thomas Mt.-9 strains
Vandeventer Flat-17 strains	Vandeventer Flat-10 strains
Santa Rosa Mt.-14 strains	Santa Rosa Mt.-5 strains
Piñon Flat-31 strains	Piñon Flat-18 strains
Mather-9 strains	Mather-8 strains

Arrowhead gene arrangement

Keen Camp-11 strains
Thomas Mt.-5 strains
Vandeventer Flat-7 strains
Santa Rosa Mt.-7 strains
Piñon Flat-13 strains

Treeline gene arrangement

Piñon Flat-1 strain

Pikes Peak gene arrangement

Santa Rosa Mt.-1 strain

Wild Strains-Mather background. Third chromosomes from Piñon Flat and Keen Camp transferred to a Mather background of X, II, IV, and V chromosomes. (Don Mitchell)

Piñon Standard III, Mather X, II, IV, V - 9 strains
Piñon Chiricahua III, Mather X, II, IV, V - 8 strains
Keen Camp Standard III, Mather X, II, IV, V - 9 strains
Keen Camp Chiricahua III, Mather X, II, IV, V - 8 strains

Lethal Strains. Nonallelic third-chromosome lethals derived from wild males. Balanced over Lb or, In (Santa Cruz)/lethal, In(Cuernavaca). New strains being added. (R. H. T. Mattoni)

Piñon Flat - 8 strains
Vandeventer Flat - 7 strains

Mutants

Chromosome II

1 gl
2 up bx Ba gl/lethal, In

Chromosome III

3 or
4 pr
5 or pr
6 or Sc pr cv
7 or Bl Sc pr/lethal, In (Cuernavaca)
8 Lb or, In (Santa Cruz/lethal, In (Cuernavaca)

Chromosome IV

9 in j hk Cy/in j hk

Multichromosomal

10 Ba or Cy

New mutants, not listed above, derived from both wild and irradiated stocks will be reported later. (F. Ball)

MINNEAPOLIS, MINNESOTA: UNIVERSITY OF MINNESOTA, DEPARTMENT OF ZOOLOGY

D. pseudoobscura
D. funebris
D. gibberosa

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY, OSBORN ZOOLOGICAL LABORATORY

D. americana americana (Independence)
D. americana americana (Western)
D. americana texana (Florida)
D. ananassae (Cristobal)
D. biopaca (Guatamala)
D. busckii (Honolulu)
D. cordata (Quirigua)
D. equinoxialis (Teffe)
D. funebris (Rexburg, Idaho, 2)
D. funebris (Stockholm)
D. funebris (white of Kiil)
D. gibberosa (S. Mexico)
D. hydei (Yucatan-4)
D. hydei (N x vr, Spencer)
D. lacicola (Fairbank, Minn.)
D. melanica (Walnut C)
D. mohavensis (Choc. Mts.)
D. montana (Cottonwood Canyon)
D. nebulosa (Georgetown)
D. novamexicana
D. paramelanica (S. Amherst)
D. repleta (New Haven)
D. sellata (Chilpancingo)
D. simulans (S. Africa)
D. spinotheca
D. takahashii (Formosa)
D. virilis (Japan)
D. willistoni (Quirigua)
D. yampa (Colorado)

NEW LONDON, CONNECTICUT: CONNECTICUT COLLEGE

D. gibberosa

NEW YORK, NEW YORK: COLUMBIA UNIVERSITY

D. persimilis

Wild: 16 strains

Mutants

or Cy
Delta

D. prosaltans

Wild: 3 strains

Chromosome 1

1 wy/sc
2 Ro/transl Y-II
3 s m ct/fa/transl I-II
4 w m y/fa/transl I-II
5 m y se
6 sd y se
7 g

Chromosome 2

1 Pm Belem
2 Pm Guatemala
3 Gf Pm
4 Bd Cy
5 L px
6 px
7 grv
8 Pm S Cy/inversion/lethal
10 En px

Chromosome 3

1 Delta ar/inversion/lethal
2 ar

D. pseudoobscura

Wild strains (geographical): 33 strains

Wild strains homozygous for different gene arrangements in the third chromosome:

Piñon, California:
Standard--12 strains
Chiricahua--12 strains
Arrowhead--11 strains

Mexico:
Chiricahua--12 strains

Wild strains collected in 1950:
Mather, California--12 strains
Lehman Cave, Nevada--14 strains
Mono Lake, California--28 strains

Chromosome 1

1 Pt w^e mg
2 y sn v co sh
3 sex ratio/y sn v co sh
4 Homoterminal S R

Chromosome 2

5 ca
6 gl
7 up bx Ba gl(inversion)/lethal

Chromosome 3

8 or
9 or pr
10 or px
11 or Sc pr cr
12 or Bl Sc pr(Standard)/lethal Cuernavaca
13 or Bl Sc pr(Standard)/ro
14 Em/intersex I
15 Em/intersex II
16 Lob or (Santa Cruz)/lethal Cuernavaca

Chromosome 4

17 in hk j Cy/in hk j

Multichromosomal

18 Ba or Cy

D. willistoni

Wild: 28 strains

Chromosome 1

1 sc Inc se re(Belem)/y(Rio)
2 w^e sc Inc ru(Belem)/y(Rio)
3 w^e loz pn(Belem)
4 co/pn
5 sn y pn ru(Belem)
6 w sc ct pn (red st)
7 m

Chromosome 2

1 S Hk abb bw/Em(inversion)
2 S Hk abb bv(inversion 207/lethal
3 abb bw
Also 9 unstudied recessive mutants on Chromosome 2

Chromosome 3

1 Delta Ri (pink)
2 Delta pink(inversion 133)/lethal
3 Rf
4 ld bx
5 Delta Rf/ss^a
Also 5 unstudied mutants on Chromosome 3

Other Species

D. athabasca
D. azteca (2 strains)
D. bocainensis
D. capricorni
D. cardinoides
D. duncani
D. equinoxialis
D. fumosa
D. gibberosa
D. miranda
D. mirim
D. montana
D. montium
D. neocardini
D. paulistorum
D. sturtevanti (2 strains
D. tropicalis (2 strains)

OAK RIDGE, TENNESSEE: OAK RIDGE NATIONAL LABORATORY, BIOLOGY DIVISION

D. virilis

Wild Strain: Pasadena

Chromosome 1

v^{48b}
mt^7
y^{40a}

Chromosome 2

va

Chromosome 3

sv

Chromosome 4

cd

Chromosome 5

pe
pe^{Jap}
B^4
a
st pe^{Jap}
B^4 pe^{Jap}
st B^3
ru pe^{Jap}
es pe^{Jap}
es B^3
st mh
st es pe^{Jap}
ru st pe^{Jap}

Chromosome 6

gl

Chromosomes 1 and 5

v^{48a}; pe
v^{48a}; es pe

Chromosomes 2 and 5

b; pe^{Jap}

Chromosomes 4 and 5

cd; es pe

Chromosomes 2, 3, and 5

b; t; cd; pe^{Jap}

Chromosomes 2, 3, 4, and 5

b; t; cd; pe^{Jap}
b; tb gp; cd; pe^{Jap}

PHILADELPHIA, PENNSYLVANIA: INSTITUTE FOR CANCER RESEARCH, and LANKENAU HOSPITAL RESEARCH INSTITUTE

D. funebris

m1 +A
m2 +B
m3 w (from +B)

D. pseudoobscura

n1 Pw^{HR} mg^2 s
n2 Pw^e mg^2 s
n3 Ssc/sr l Y & Pw^e mg^2 s

D. simulans

o1 + South Africa
o2 net pm (b, py, sd)

ST. LOUIS, MISSOURI: WASHINGTON UNIVERSITY, DEPARTMENT OF ZOOLOGY

D. robusta

Chromosome 1

vermilion echinus

Chromosome 2

cardinal acrostichal

Chromosome 3

Scarlet-D

Wild-type: 10 strains

Other Species

D. americana (3 strains)
D. cardini (8 strains)
D. neocardini
D. polymorpha
D. simulans
D. tripunctata (10 strains)
D. cardinoides (2 strains)

SALT LAKE CITY, UTAH: UNIVERSITY OF UTAH

S. simulans: v

UPTON, L. I., NEW YORK: BROOKHAVEN NATIONAL LABORATORY, BIOLOGY DEPARTMENT

D. americana (Independence)
D. ananassae (Cristobal)
D. busckii
D. equinoxialis (Teffe)
D. funebris
D. hydei (Yucatan-4)
D. nebulosa (Georgetown)
D. novamexicana
D. persimilis
D. pseudoobscura
D. repleta
D. simulans (South Africa)
D. takahashii
D. texana (Florida)
D. virilis (Japan)
D. willistoni (Quirigua)
Zaprionus vitiger (South Africa)

AUSTRIA

Vienna: Institut f. allgemeine Biologie d. Universität

Note: Stock list unchanged. See DIS-24, p. 69.

BRAZIL

Curitiba, Paraná: Universidade do Paraná, Laboratory of Genetics

D. ananassae: Morretes; Teixeira Soares; Guaratuba; Lapa; Porto da Passagem; Ponto Fiscal (state of Paraná), and Recife (state of Pernambuco).
D. auraria: Hangchow, China.
D. melanogaster: mutants.
D. montium: Morretes, light and polymorphic stocks; Pinheiral (state of Rio de Janeiro), dark form; and Honolulu, Hawaii, light form.
D. nebulosa: Del Rio, Texas; and Lapa (state of Paraná).
D. paulistorum: Vila Atlântica (state of São Paulo).
D. simulans: Lapa; and Curitiba (state of Paraná).
D. tropicalis: Palma (state of Goiás).
D. willistoni: Monjolinho (state of Goiás).

São Paulo: Universidade de São Paulo, Dept. de Biologia Geral

D. ananassae: Doleschall, 1858, São Paulo (State of São Paulo)
D. annulimana: Duda, 1925, Vila Atlântica (State of São Paulo)
D. araicas: Pavan and Nacrur, 1950, Belem (State of Pará)
D. arauna: Pavan and Nacrur, 1950, Pirassununga (State of São Paulo)
D. bandeirantorum: Dobzhansky and Pavan, 1943, Pirassununga and Mogi das Cruzes (State of São Paulo)
D. bocainensis: Pavan and Cunha, 1947, Pirassununga and Mogi das Cruzes
D. capricorni: Dobzhansky and Pavan, 1943, Mogi das Cruzes
D. cardinoides: Dobzhansky and Pavan, 1943, Mogi das Cruzes
D. guaramunú: Dobzhansky and Pavan, 1943, Mogi das Cruzes
D. mediopunctata: Dobzhansky and Pavan, 1943, Reuter (State of Rio Grande do Sul), Mogi das Cruzes (State of São Paulo), Lambedor (State of Paraná)
D. mercatorum mercatorum: Patterson and Wheeler, 1942, Sanat Barbara, California; S. José da Costa Rica; Lima, Peru
D. mercatorum pararepleta: Dobzhansky and Pavan, 1943, strains from 40 localities of Brazil; strain from Santa Cruz de la Sierra
D. meridiana rioensis: Patterson, 1943, Atlixio, Mexico
D. mesofragmatica: Duda, 1925, Campos do Jordão (State of São Paulo)
D. montium: Honolulu, Hawaii; Pirassununga, Itanhaem, São Paulo; Lambedor (State of Paraná)
D. neocardini: Streisinger, 1946, Pirassununga (State of São Paulo)
D. neoelliptica: Pavan and Magalhães, 1950, Anápolis (State of São Paulo)
D. neosaltans: Pavan and Magalhães, 1950, Mogi das Cruzes (State of São Paulo)
D. paranaensis: Barros, 1950, Capitão Heitor Port, Paraná river; Cataratas de Iguassu; Foz do Iguassú; Pirassununga (State of São Paulo); Belém (State of Pará); Imperatriz (State of Maranhão); Santo Angelo (State of Rio Grande do Sul).
D. peninsularis: Patterson and Wheeler, Lake McKethan, Florida; Tarpon Springs, Florida
D. polymorpha: Dobzhansky and Pavan, 1943, dark selected strain from Prata (State of São Paulo)
D. pulla: Pavan and Cunha, 1947, Mogi das Cruzes (State of São Paulo)
Drosophila species 1, Medio group: São Paulo
Drosophila species 2, Dreyfus group: Vila Atlântica (State of São Paulo)

D. sturtevanti: Duda, 1925, Belém do Pará (State of Pará), Vila Atlântica and Pirassununga (State of São Paulo), Palmares (State of Goiáz), Imperatriz (State of Maranhão)
D. willistoni: Sturtevant, 1921, 25 strains from different parts of Brazil
D. willistoni:

Mutants

Chromosome 2: Emarginate/purple-hairless
Chromosome 2: Star-Hooked-abbreviated brown Inv. 207/broad
Chromosome 10: white

Balanced stock S Hk abb bw Inv. 207/lethal

Numbers of different lethals	Locality	State
28	Catuni	Bahia
25	Vila Atlântica	S. Paulo
40	Mogi das Cruzes	S. Paulo
6	Pirassununga	S. Paulo
5	Palmares	S. Paulo
10	Cruzeiros do Sul	Territory of Acre
5	Japiim	Territory of Acre
1	Mucajai	Amazonas
5	Rio Negro	Amazonas
2	Belém	Pará
2	Palmas	Goiáz

GERMANY

Berlin-Buch: Institut für Medizin und Biologie, Genetische Abteilung

D. funebris

Wild Stocks

90 normal
91 normal (Buch, 1947)

X Chromosome

92 ev
93 w
94 y
95 yw

Autosomes

96 Pch
97 st

Other Species (wild)

98 D. busckii
99 D. hydei
100 D. repleta
101 D. simulans - v
102 D. virilis

Berlin-Dahlem: Institut für Genetik der Freien Universität Berlin

D. busckii
D. funebris
D. hydei
D. pseudoobscura (Cuernavaca)
D. repleta
D. simulans
D. virilis

Göttingen: Max Planck-Gesellschaft, Institut für Tierzucht und Tierernährung

D. virilis

1 +
2 ac gl
3 b;t;cd;es
4 b;tb gp;cd;pe
5 va;gl
6 y^{40a}
7 y^{40a};b;cd;es

D. immigrans
D. pseudoobscura A (Texas)
D. repleta
D. simulans (v)

Göttingen: Zoologisches Institut der Universität

D. funebris

1 +
2 M
3 Pch

GREAT BRITAIN

Edinburgh, Scotland: Edinburgh University, Institute of Animal Genetics

D. ambigua (2 strains)
  1 wing-vein mutant (untested)
D. ananassae
D. busckii
D. funebris
D. guyenoti
D. immigrans
D. littoralis
D. miranda
D. montium
D. obscuroides
D. persimilis
D. pseudoobscura
D. simulans (4 strains)
D. subobscura (8 strains)
D. transversa
D. tristis
D. virilis
D. sp. (repleta group)

Harpenden, Herts: Rothamsted Experimental Station

D. funebris

London, England: University College, Department of Biometry

D. persimilis

Wild-type

Aspen I
Tuolumne 10
Tuolumne 11

Mutants

sn bu

D. simulans

Aust Tex +
st pe
y w

D. pseudoobscura

Wild-type

Aspen 3
La Grande
Santa Barbara I
Texas

Mutants

P tb
tb b v se sp
w
se ll sp tt

D. subobscura

Wild-type

+ KÜSSNACHT

X Chromosome

Standard order

c(X) cv sc
v w +/v w^{co} sc
ct^{frM} be
ct cv^2
ct y^2
Bx^2
da oc
oc (fs)
v N^5 rr w^{co} sc
ct^{an}
sn v cv sc
ry v (ct)
m ct bnt v sc
ct^1/ct^{frM} bnt
pm ct sn cp
scl wi
bnz v cv
w^a sc
pm ct/ry ct
v w/v w^{co} y wi
ct^k (segregating for ? In(X)3)
m ct sn cp v sc

Inverted orders

bnz^2 v In(X)1+2
+ In(X)1+2
ct cp sn v In(X)1+2
ct cp sn v y In(X)1
sn cp In(X)3

Balbiani Ring Chromosome

Standard order

th ma
s : e
ma int
log th int
sth int ey wt
wn th int
sepia
s^2 (order unknown)

Inverted order

s th ma int
ma mop

Chromosome 3

ho
r
nt
pf nt
pp; segregating for gs

Chromosome 4

pl pp op
pl pp pt
otp pp fs
pt^c
sj pl pp fs
sj pl otp pp

Chromosome 5

ch cu Dl
ch for Ba
ar
Va'
Va^x
Ba for st
Ba for rn
Ba for ant
Ba for ix

Mixed Stocks

ma; pl pp
ma: ch

Unplaced Autosomal Loci

kk^2
cm
csp:int
Fh
pa
dk
forked-Milani (ma int)

ITALY

Pavia: Università, Istituto di Genetica

D. acanthoptera (1 strain)
D. affinis (4 strains)
D. algonquin (4)
D. ambigua (3)
D. athabasca (1)
D. azteca (1)
D. baeomyia (1)
D. bifasciata
  Wild stocks (2)
  Mutants: f, ob, or
D. busckii (1)
D. buzzatii (3)
D. cardini (1)
D. duncani (1)
D. funebris (1)
D. gibberosa (1)
D. guttifera (1)
D. helvetica (1)
D. hydei (1)
D. immigrans (1)
D. kuntzei (1)
D. lebanonensis (1)
D. littoralis (1)
D. miranda (1)
D. mirim (2)
D. narragansett (1)
D. nitens
  Wild stocks (2)
  Mutant: or
D. obscuroides (5)
D. pallida (1)
D. persimilis (3)
D. polychaeta (1)
D. prosaltans (1)
D. pseudoobscura (7)
D. putrida (1)
D. repleta (1)
D. robusta (1)
D. sellata (1)
D. simulans (2)
D. spinofemora (1)
D. subbadia (1)
D. subobscura
  Wild stocks (24)
  Homozygous standard
  (2): Esperöd
  Küssnacht
D. testacea (1)
D. transversa (1)
D. tripunctata (1)
D. tristis (1)
D. victoria (1)
D. virilis (1)
D. willistoni (1)

Pavia: Università, Istituto di Zoologia dell'Università e Centro di Genetica del Consiglio Nazionale Ricerche

D. affinis (1 strain)
D. ambigua (1)
D. bifasciata (1)
D. obscuroides (1)
D. persimilis (1)
D. pseudoobscura (1)
D. simulans (1)
D. subobscura (5)
  Mutant: unlocated
  autosomal-vg^{48j}

JAPAN

Kyoto: Kyoto University, Zoological Institute

D. ananassae

Wild Stocks

1 Baton Rouge
2 Campus Oahu, Hawaii
3 Louisiana
4 Mampa, Africa
5 Porto Rico
6 Sao Paulo
7 Texas

Mutants

8 Bn (Broken)
9 brown eye
10 cd f
11 cd f bw
12 cd f ru^2
13 px d
14 ru (roughoid)
15 y

D. virilis

Chromosome 1

1 bb
2 buff
3 cv mt w^e sb
4 si^{16}
5 v
6 y
7 y^5 $cv(f^4)$ ap

Chromosome 2

8 C^{21}
9 R

Chromosome 3

10 cn
11 $N3^a$

Chromosome 4

12 cd
13 px

Chromosome 5

14 B^4
15 es pe
16 pe
17 po
18 Sb
19 st
20 st B^3 pe (es)
21 Sv po
22 Sv^2

Chromosome 6

23 ac gl
24 hp
25 ski hp/Gp gl

Multichromosomal

26 b bk dt
27 b t^2 cd ni (es pi)
28 cn px pe
29 es px
30 px B^3 pe
31 R (tb) gp cd (pe)
32 w ds

Inversion

33 In(X) Spd

Translocation

34 T (X-4)
35 T (Y-3)

Wild Stocks

America (3 strains)
Japan (ca. 10 strains)

Other Species

D. auraria (1 strain)
D. hydei (1 strain)
D. montium (1 strain)
D. simulans (1 strain)
D. takahashii (2 strains)
D. texana
D. willistoni (2 strains)

Misima, Sizuoka-ken: National Institute of Genetics

D. ananassae: Campus Oahu, Hawaii
Mampa, Africa
D. hydei: Lebanon
D. simulans: Ennis, Texas
D. virilis: E-Takasu, Hokkaido
Tokyo
D. willistoni: Standard

Osaka: Osaka University, Genetical Laboratory

D. virilis

Wild Stocks

1-3 wild types in America
4-18 wild types in Japan

Chromosome 1

19 bb
20 cv mt w^e sb
21 mt Bx w
22 sd
23 si^{16}
24 v^4 si^{16}
25 w si^{16}
26 y ap
27 y cv ap

Chromosome 2

26 eb
27 ro2b

Chromosome 3

28 rs

Chromosome 5

29 es
30 es pe
31 Sb
32 Sv po

Chromosome 6

33 hp
34 ski hp/Gp gl

Multichromosomal

35 si; b; t^2; cd; es
36 cv; cd
37 cn; px; pe
38 R; tb; gp; cd; pe
39 st; B^3; pe

Inversion

40 In(X)Spd

Translocation

41 T(X-4)

Virilis Group

D. americana americana (1 strain)
D. americana texana (2 strains)
D. novamexicana (1 strain)

Other Species

D. ananassae (2 strains)
D. auraria (4 strains)
D. busckii (1 strain)
D. funebris (1 strain)
D. hydei (3 strains)
D. immigrans (6 strains)
D. lutea (7 strains)
D. montium (3 strains)
D. simulans (1 strain)

Sapporo: Hokkaido University, Institute of Zoology

D. auraria: Sapporo, Otaru, Shioya, Muroran, Imagane, and Higashi-Takasu
D. busckii: Sapporo, Otaru, and Imagane
D. funebris: Otaru, Sapporo, and Higashi-Takasu
D. immigrans: Sapporo, Otaru, Akkeshi, and Samani
D. repleta: Shioya
D. robusta group: Sapporo, and Imagane
D. suzukii: Shioya, and Imagane
D. transversa: Sapporo, Otaru, Shioya, and Imagane
D. virilis: Sapporo, Otaru, Shioya, and Nakashibetsu

Tokyo: Tokyo Metropolitan University, Department of Biology

D. ananassae

Wild Stocks

1 Texas
2 TL_1
3 TL_3
4 TL_4
5 TL_{3-4}
6 TL_{3-11}
7 Barro Colorado, Panama-55
8 Barro Colorado, Panama-69
9 Barro Colorado, Panama-74
10 Turrialba, Costa Rica-101
11 Turrialba, Costa Rica-104
12 Turrialba, Costa Rica-116
13 Turrialba, Costa Rica-125
14 São Paulo
15 Mampa, Africa
16 Monterrey, Mexico
17 Christobal, Mexico
18 Campus Oahu, Hawaii
19 Porto Rico
20 Baton Rouge, La.
21 Hawaii-H
22 H^A(Hawaii)
23 H^B(Hawaii)
24 M^A(Monterrey)

Mutants

25 Ax
26 Ax/ds
27 bb(bb-IV?)
28 Bn
29 Bn-c
30 Bn-c bw
31 bw
32 cd f
33 cd f bw
34 cd f bw ru_{ec}
35 cd f bw sk
36 cd f ru_{ec}
37 cd cv-a
38 cv-a
39 dpl

40 f
41 pxd
42 ru
43 ss-HH
44 ss-HX
45 trd
46 y
47 y f

Other Species

D. americana americana (wild, 1 strain)
D. busckii (wild, 3 strains)
D. funebris (wild, 1 strain)
D. hayashii (wild, 2 strains)
D. hydei (wild, 2 strains)
D. immigrans (wild, 5 strains)
D. lebanonensis-like (wild, 1 strain)
D. lutea (wild, 2 strains)
D. macrospina (wild, 1 strain)
D. montium (wild, 2 strains)
D. nigromelanica-like (wild, 1 strain)
D. novamexicana (wild, 1 strain)
D. obscura-like (wild, 2 strains)
D. pseudoobscura (wild, 2 strains; mutant, 1 strain)
D. rufa (wild, 2 strains)
D. simulans (wild, 1 strain)
D. sordidula (wild, 1 strain)
D. suzukii (wild, 1 strain)
D. takahashii (wild, 2 strains)
D. transversa (wild, 3 strains)
D. virilis (wild, 7 strains; mutant, 3 strains)
D. willistoni (wild, 1 strain; mutant, 1 strain)

Tokyo: University of Tokyo, Botanical Institute, Laboratory of Genetics

D. ananassae
D. auraria
D. busckii
D. hydei
D. montium
D. suzukii
D. virilis

SOUTH AFRICA

Johannesburg: University of the Witwatersrand, Department of Zoology

D. persimilis

1 Sequoia
2 or
3 or Cy

D. pseudoobscura

Wild Stocks

1 Aspen
2 Texas

Chromosome 1

3 se
4 tb b v se sp
5 v

Chromosome 3

6 or
7 or pr
8 pr

D. simulans

Wild Stocks

1 Johannesburg
2 West Rand
3 Mkuzi Game Reserve

Mutants

4 b

D. subobscura

1 Wild strain
2 v w/v w^{co}
3 se
4 pm cf bg^2 cn cp
5 ma; pl pp

Other Species

1 D. funebris (Johannesburg)
2 D. miranda
3 D. montium
4 D. opisthomelaina (Nelspruit and Mkuzi Game Rserve)
5 Zaprionus tuberculatus (Zoutpansberg)
6 Z. vittiger (Johannesburg and Mkuzi Game Reserve)

SPAIN

Barcelona: Universidad de Barcelona, Centro de Genética Animal y Humana

D. bifasciata (Pavia)
D. busckii (Barcelona)
D. funebris (3 strains: Asteggio, Barcelona, Lérida)
D. immigrans (Barcelona)
D. repleta (Barcelona)
D. simulans (Barcelona; mutant stocks)
D. subobscura (5 strains from Spain; mutant stocks)
D. testacea (Montnegre)
D. transversa (Montnegre)
Scaptomyza graminis (2 strains from Spain)

NEW MUTANTS

D. ambigua

Report of F. Mainx

Bd: Beaded Karlik. Autosomal, dominant. Like Beaded in D. melanogaster. Most variable. Lethal when homozygous.

D. bifasciata

Report of A. A. Buzzati-Traverso

f: forked Peschiera, Nov. 1950. Sex-linked recessive. Bristles shortened, bent, with ends split or bent sharply; similar to forked of D. melanogaster. Found in the Pavia strain.

ob: occhio bruno Peschiera, summer 1949. Autosomal recessive. Eye color dark and dull; similar to oo of D. melanogaster. Found in progeny of X-rayed male.

or: occhio rosso Peschiera, spring 1949. Autosomal recessive. Eye color bright red; similar to cn of D. melanogaster. Found in the Pavia strains.

D. pseudoobscura

Report of Etta Käfer

Wi: Winkel 1-125 ± 5. Isolated from a stock arp/arp. Reduces the angle between main and side branches of the arista in a definite way. In wild-type flies this angle measures, on the average, 40°; in Wi/+ flies, 31°; and in Wi/Wi flies, 22°. (Pub.: E. Käfer, 1950, Arch. Jul. Klaus-Stiftung 25: 512-514.)

D. robusta

Report of M. Levitan

2L-5: (a new gene arrangement) Single-step inversion from standard (2L). Proximal break in region 22, distal break in 18. Carried by an uninseminated female collected October 1, 1950, near Blacksburg, Va.

D. subobscura

Report of J. M. Clarke

N: Notch Three putative occurrences of Notch were found in stock last year: two as single females, which were sterile (March and September, 1951); and one as five females, three with smooth eyes and two with the rough eyes associated with rr (March, 1951). This has been established in stock and called N^5.

Va: Varicose Similarly two unrelated females, autosomal dominants showing the Varicose pleotropisms, have been found in stock. One (March, 1951) has produced a stock Va^x, and the other (September 1951) only a few sterile offspring. J. Maynard Smith has found that Varicose is an allelomorph of Delta, and that the compound Va'/De is lethal, as are the homozygotes Va'/Va' and De/De.

Report of H. Spurway

ax: abdominal extras Spurway, 51b. Segregating in stock of Ba for rn. The abdominal spiracles are more difficult to see in subobscura than in other species. This autosomal recessive seems to misplace them in the membranous part of the abdomen. Perhaps for this reason they are much larger, untidily chitinized, and more conspicuous. Frequently the chitinization of the spiracle seems to be everted so that a malformed bristle is present. Difficult to score but fully penetrant. I am indebted to Miss J. M. Clarke for this interpretation of the phenotype.

ct^{K}: cut Kunze. New allelomorph at cut locus. Most extreme and constant yet found. Expression similar in males and females. The wings are pointed because of a characteristically shaped incision removing parts of the submarginal cell and the 1st and 2nd posterior cells. The 4th longitudinal vein usually does not extend to the new margin of the wing. Vibrissae present. Received from E. Kunze, Institut. f. allgemeine Biologie, Vienna.

da: date Spurway, 50l. Sex-linked recessive. Single male found in experimental paired mating. Pale brown eye and white testes, indistinguishable from copper and bronze. This is the third locus on the X chromosome producing this complex of characters, which has not yet been reported from any other member of the obscura group.

dk: dark Spurway, 50j. Segregating in stock of ct$^{fr\ M}$. Autosomal recessive. Dark eye color like plum, but with white testes.

In(X)3 Dominant inhibitor of crossing over in the X chromosome, discovered November 1950 in descendants of Barton-c fly (see ix). Professor Mainx tells me there was only one X-chromosome inversion segregating in the Barton-c stock of which he had a subculture; therefore In(X)3 is probably simple inversion 1 in the A element of the salivary-gland-chromosome map prepared by his Institut. Its genetics are: In 4425 flies from ct sn cp v sc (S)/+++++ In(X)3 females, the recombination values were as follows: ct - sn, 41.85 ± 0.74; sn - cp, 5.47 ± 0.35; cp - v, 4.42 ± 0.31; v - sc, 0. Among these flies there was one double crossover involving regions 2 and 3, a ct cp fly. If there were no interference 10.7 would have been expected, but the presence of even one is, perhaps, remarkable. The total data from v sc S/++ In(X)3 females are 4845 ++, 4683 v sc, and no recombinants. v w^{co} y S/+++ In(X)3 females have produced 840 +++, 723 v w^{co} y flies, and no recombinants. A lethal on the Standard chromosome segregated in two cultures. Bird, in 1947, found the Standard recombination values to be ct - sn, 35.6; sn - cp, 5.8; cp - v, 38.0; v - w^{co}, 29.7; v - sc, 44.7; y - sc, 0.37.

ix: intersex Five intersexes were found by Dr. R. Milani in 4 out of 21 F_2 cultures bred from the offspring of a single female recorded as Barton-c collected near Cambridge, England, early summer 1950. Sibs of these flies were presented to this laboratory. The condition is due to an autosomal recessive gene located on chromosome 5. Only XX zygotes are affected; ix/ix males are normal and fertile. Penetrance may be fairly complete, but can fall to about 50%. Expression is variable. All intersexes have some bristles of the sex combs. These are symmetrical. The ovipositor varies from normal to vestigial or a scar in the chitinized membrane of the belly. The male genitalia vary from absence, through an undifferentiated excrescence dorsal to the ovipositor but ventral to the anus, to comparative perfection but rotated clockwise. Large portions of the male internal genital apparatus have been seen, but testes never. The anus shows every gradation from female to male. What may be described as some degree of division of the dorsal lip of the "female" anus is found in almost all specimens, unlike the intersexes in D. melanogaster. A common type has two dorsal and one ventral valve to the anus, an almost perfect ovipositor, and no male genitalia. In some intermediate types the genital arch (Biology of Drosophila, ed. Demerec, p. 413) appears as tubercles in the intersegmental membrane dorsal to the anus between it and the eighth tergite (op. cit., p. 412). This implies disagreement with the homologies propounded by G. F. Ferris in the article referred to. Similarly, from our series of intersexes it is clear that the male anus is homologous with the dorsal lip of the female anus. The sex-comb-like hairs flanking the penis, which are used for classification in this genus, appear entirely separately from any plates, together with penis components stuck like pins in a teratomatous fashion in the relatively unchitinized excrescence. In more male types the different components of the male genitalia and anus become organized so that their relative positions to each other are normal but the whole structure is rotated through 45° clockwise relative to the ovipositor rudiment and the rest of the fly.

pf: puffy scutellum 3 Spurway, 50k. Scutellum twice the normal volume. Expression variable. Chitinization of the scutellum appears normal. For the linkage relationship I am indebted to Miss M. Wiener.

s^2: scarlet^2 2 Spurway, 50l. Several males and females in experimental paired mating. Darker re-occurrence of 2nd-chromosome eye color scarlet.

D. tropicalis tropicalis

Report of J. Ives Townsend, Jr.

After X-radiation of wild-type males, one autosomal dominant, two autosomal recessives and one sex-linked recessive were recovered.

D. tropicalis cubana

(a new subspecies to be described elsewhere)

Report of J. Ives Townsend, Jr.

se: sepia Townsend, 51d. Sex-linked recessive. From X-rayed wild-type male. Eye color on emergence more brownish and translucent than normal, darkening to sepia.

In additional to se, two autosomal dominants, one autosomal recessive, and one sex-linked recessive were recovered after X-radiation of wild-type males.

D. virilis

Report of William K. Baker

pe^{m1}: peach-mottled 1 Baker. 47i24. T(Y;5)pe^{m1}. From X-rayed Pasadena wild-type male. Heterozygous flies ($pe^{m1}/+$) are wild-type, pe^{m1}/pe are mottled. Hyperploid flies ($pe^{m1}/pe/pe$) are mottled, fully viable and fertile. Break in chromosome 5 is distal to pe locus, but rearrangement is not visible in salivary-gland chromosomes.

pe^{m2}: peach-mottled 2 Baker. 51b26. T(3;5)pe^{m2}. From X-rayed Pasadena wild-type male. Heterozygous flies ($pe^{m2}/+$) are wild-type, pe^{m2}/pe are mottled. Break in chromosome 5 at or close to centromere;

break in chromosome 3 in the 3D1 region (Griffen's map).

pe^{m3}: peach-mottled 3 Baker. 51c2. T(4;5)pe^{m3}. From X-rayed Pasadena wild-type male. Heterozygous flies ($pe^{m3}/+$) are wild-type, pe^{m3}/pe are mottled. No rearrangement in salivary chromosomes is visible. Assume both chromosomes 4 and 5 are broken close to centromere.

pe^{m4}: peach-mottled 4 Baker. 51c30. T(3;5)pe^{m4}. From X-rayed Pasadena wild-type male. Heterozygous flies ($pe^{m4}/+$) are wild-type, pe^{m4}/pe are mottled. Chromosome 5 apparently broken at centromere, chromosome 3 broken close to 3B2C (Griffen's map).

Report of Iben Browning

A mutation that occurred spontaneously in November, 1950, at our laboratories involves the eosin (w^e) locus of the X chromosome of D. virilis. It appears eosin, and is indistinguishable from the eosin (w^e) stock carried by the Genetics Laboratory of the University of Texas at Austin.

Report of T. Imaizumi

Confluent^{21} 1949. X-ray-induced from wild stock (Kitakata-24). Typical confluent, homozygous embryonic lethal. Lethal phenotype is imperfect contraction of the germ band.

Research Notes

Anders, Georges Pleiotropic effect of lozenge clawless.

The lozenge-clawless (lz^{cl}) mutant which affects the size, color, and structure of the eye, and implicates reduction of claws in both sexes and in females the loss of parovaria and spermathecae, also effects a considerable reduction of the third antennal segment. Histologically a certain type of thin-walled blunt hairs (possibly sensilla basiconica) were found mostly lacking. More accurate investigations involving differences between the alleles lz^{cl}, lz^{s}, lz^{34k}, lz^{46f24} and lz^{g} and their compounds are in course.

Anders, Georges Pleiotropic pattern of the lozenge-mutant in D. pseudoobscura.

Investigations on pseudoobscura lozenge showed the pleiotropic pattern to be very similar to that of melanogaster. The eyes, claws, and antennae are affected by reduction. In females the spermathecae and parovaria are lacking. Owing to this great similarity in the pattern of gene action a homology of these genes can be postulated.

Asahina, Kazuo Studies on the Taxis of D. melanogaster.

It seems highly probable that the behavior of D. melanogaster is controlled by the phenomena of four fundamental taxises: phototaxis, chromotaxis, negative geotaxis, and negative aggrotaxis. In this investigation the relation between these taxises and behavior was studied. The experiment on chemotaxis, one of the important factors responsible for the matter, has not yet been performed. The results obtained in experiments in which temperature was 25° C are as follows.

1. Phototaxis (investigated by light within the limits of 1,000 Lux.): Males and females show similar positive responses. Response of taxis becomes stronger in accordance with intensity of stimulus of the light, and it reaches a constant level after 30 minutes of irradiation. The animals also show strong responses under green color, the complementary color of red, and white illumination, whereas a weak response is observed under yellow color, the complement of blue. But under orange illumination they show a negative response.

2. Chromotaxis: In general, the taxis appears strongly by the light of long wave and weak by short wave. It appears also strong by green and weak by yellow. This taxis is similar in males and females, and also shows constant reaction after 30 minutes of irradiation.

3. Negative Geotaxis: The flies begin to fly up lively by the illumination of every color when they are changed an angle from horizon. This reaction becomes maximum during the first 5 minutes and decreases afterwards. The animals show the strongest responses under green light at 45° and under red at 90°, but do not show any difference under other colors.

4. Negative Aggrotaxis: It is observed in test tube that the individuals migrate from dense part to that of scattered under the illumination of every color. This phenomenon is increased under light of red, green, and orange, while it is comparatively weak under blue and yellow light. These responses appear considerably quick and both males and females show the same reaction. These experiments show that the similar inclination exists between the cooperative effects of green and red and those of yellow and blue. The behavior of the flies, therefore, does not always consist with the approximation of the wave length and this phenomenon is worthy of note.

The present author is now investigating the interrelation of intensity of these taxises, and that between the taxis and ovulation or sex ratio.

Basden, E. B. Drosophila in Scotland. During 1950-51 a survey of the Drosophila fauna of Scotland produced the total of 18 species listed below. Those marked with an asterisk are new to the British list, while some of the others are recorded for the first time from Scotland.

1. D. subobscura Collin. Common and widespread. An out-of-doors species that only exceptionally exters buildings. It is most plentiful in and near woodlands, though it is the only species that can be expected in wide open spaces remote from trees (moorlands, etc.).

2. D. obscuroides Pom. Widespread but not as frequent as (1). Does not enter buildings, and keeps more to wooded areas.

3. D. tristis Fall. Has a widely scattered distribution but is only infrequently met with. My most northerly record is Lat. 57° 53'.

4. D. ambigua Pom. Found as yet at only 4 or 5 places up to Lat. 57° 36'.

*5. D. sp. nr obscuroides (New species.) Has two long bristles on each ovipositor plate. Quite common in 1951 at apple baits. Not yet caught north of Lat. 57° 56'. This is a purely woodland species. Adults soon die in the laboratory unless fed with honey water. Many attempts to maintain stocks have failed, though ones and twos can be reared on Drosophila medium and fermenting fruits.

*6. D. guyenoti Burla. Fair numbers are attracted to apple baits, but it is not a common species and has not been trapped north of Lat. 57°.

7. D. funebris Fabr. This is the commonest species inside buildings, though it is also plentiful outdoors, but so far has not been met with north of Lat. 57° 36'.

8. D. busckii Coq. As yet found occasionally in the eastern part of Scotland up to Lat. 56° 44'.

9. D. melanogaster Meig. This will almost certainly be found in many more built-up areas but so far has not been caught by me above Lat. 56° 34'.

*10. D. simulans Sturt. This has been found on two occasions, in 1950 at Liberton, near Edinburgh, and in 1951 at Inverkeithing, across the Firth of Forth from Edinburgh.

11. D. phalerata Meig. This in the commonest toadstool species but occurs frequently at apple baits. It will probably be found wherever the larger fungi occur, but the most northerly record at the moment is Lat. 57° 36'. This and the next two species (12, 13) breed but reluctantly on Drosophila medium.

12. D. transversa Fall. This can be bred from various ground fungi in large numbers but is very rarely attracted to fruit baits, even outside the fungus season (July-October). It has not yet been found north of Lat. 56° 26'.

13. D. pallida Ztt. (= unistriata Strobl, teste Duda 1935) The eggs of this species can often be found embedded in the flesh of fungi. The adults will go to apple bait during the winter months. I have not yet found it beyond Lat. 56° 18'.

14. D. immigrans Sturt. This occurs commonly in a fruit store in Edinburgh and will doubtless occur in similar places elsewhere in Scotland.

15. D. sp. (repleta group, near bifurca P&W). This has been collected in a house in Brechin, Angus.

16. D. fenestrarum Fall. This has been caught a few times around Edinburgh.

17. D. (Parascaptomyza) disticha Duda. A few specimens have been found in windows and amongst garden herbage in the southern part of Scotland. It will breed copiously in Drosophila medium.

18. D. sp. One specimen at apple bait at Coldbackie, in the extreme north.

Bird, Myrtle J. Chemical mutagenesis.

Compounds shown in the table have been tested for mutagenic action in *D. melanogaster*. All were dissolved in 0.4% saline and injected into the abdomen of 1-2-day-old males. The determining factor for the selection of the concentration administered is the solubility of the compound. In all cases fully saturated solutions were used. In some experiments different concentrations were also tested for purposes of comparison. After treatment the males were tested for sex-linked recessive lethals by the Muller-5 technique.

Compound	Concentration used	No. chromosomes tested	No. lethals	% lethals
1:3-dimethanesulphonoxy-propane	0.2	976	30	3.1
	1.1	956	99	10.4
	2.2	624	60	9.6
1:4-dimethanesulphonoxy-butane	0.04	407	2	0.5
1:4-dimethanesulphonoxy-but-2-yne	0.1	547	30	5.5
	<0.1*	1082	47	4.3
cis-1:4-dimethanesulphonoxy-but-2-ene	0.06	1365	3	0.2
	0.2	507	4	0.8
	0.25	475	5	1.1
trans-1:4-dimethanesulphonoxy-but-2-ene	0.06	1374	17	1.2
n-butylmethane-sulphonate	0.2	707	6	0.8
Controls (combined data)	-	2449	3	0.1

* A little of compound out of solution at time of injection.

Detailed cytogenetic analysis of lethals induced by the various chemical mutagens investigated is being undertaken by Dr. Onsy G. Fahmy and will be published elsewhere.

Brunetto, Anna, and Frumento, Luigia. Salivary chromosomes of *D. ambigua*.

The salivary chromosomes of *D. ambigua* (*obscura* group) show in wild populations several heterozygous inversions. As a preliminary for physiological and population genetics researches, we have studied the salivary chromosomes of laboratory stocks, giving a map of the homozygous and heterozygous conditions. The map will be published shortly in *Scientia Genetica*. The chromosome complement of *ambigua* (A. Buzzati, 1943) is formed of two big and two small mediocentric and one point chromosomes. According to that, 8 major elements can be found in the salivary cells. The chromocenter is large and well differentiated. When broken by pressure, the limbs of the individual chromosomes tend to keep together, joined by the centromere. The sex chromosome, which in the mitotic nuclei is one of the two larger V-shaped ones, does not show in the salivaries very long arms, but they are connected by most of the chromocenter.

In our map we have adopted a nomenclature system based on letters. Dr. Prevosti (1950, Genetica Hiberica) uses numbers in his study of the salivary chromosomes of the European species of the *obscura* group. The correspondence of the chromosomes in the two systems is as follows:

Chrom.	Br.Fr.map elements		Prevosti map elements	
A	a	a'	4'	4
B	b	b'	1	1'
C	c	c'	2	2'
D	d'	d	3	3'

In our material (wild strain from Terminillo, kept in captivity for two years) we found several heterozygous inversions:

Elements	Inversions	Incidence (80 larvae obser.)
b	2 median (tandem)	40
b'	1 median; 1 subterm.	48; 40
c	1 submedian (the two chromatids can be asynaptic until the proximal end)	40
c'	none observed	
sex chr. (d (d'	none observed	
a	none observed	
a'	none observed	

The inverted homozygous order has been observed only for element c.

We have had the opportunity of studying a series of permanent preparations of salivary chromosomes of larvae of the first generation which that stock had in captivity (July-August, 1949); we find all and only the inversions which are still present in the stock. However, a few small heterozygous deficiencies have been lost. The deficiencies which have been seen in the material of the first captive generation have the following distribution: element c, one subterminal deficiency; element b', one subterminal deficiency; element a, one subproximal deficiency.

Burdette, Walter J. Incidence of tumors in different strains of Drosophila.

It is customary to classify inbred strains of mice used in cancer research according to their degree of susceptibility to spontaneous and induced tumors. Tumors appear with characteristic incidence in Drosophila strains as well. Although the number of tumors appearing is known to be influenced by culture conditions (temperature, nutrition, etc.), the incidence remains within certain limits for each stock under ordinary culture conditions. The percentage of tumorous individuals has been determined in a number of stocks for 7 to 15 generations, and some of this information is presented in the table below, with the thought that it may be useful for other investigators to have the comparative incidence of a number of tumors under the usual conditions in one laboratory. The lowered incidence of tumors in three strains after they had been made isogenic will be noted. It is apparent that a wide spectrum of tumor penetrance is available for study in Drosophila.

Incidence of Tumors in Drosophila

Stock	Months counted	Times counted	Total no. counted	Total tumorous	Per cent tumorous		
					Total	Males	Females
bw st tu	4	7	4426	24	.54	.26	.84
tu^{36a} (isogenic)	8	13	7473	222	2.97	3.21	2.73
tu^{36a}	5	9	3394	182	5.36	5.10	5.66
ed Su^{2}-dx	4	7	4022	385	9.57	11.59	7.52
f$^{257-19}$ B/In AM	4	7	2449	416	16.99	15.53	17.87
tu^{wps}	8	13	8077	1423	17.62	12.10	23.18
w^{bf} f^{5}	4	7	2827	715	25.29	30.20	19.85
lz^{3} f	8	13	1016	2428	23.88	28.53	18.59
tu^{50d}	8	15	7144	1901	26.61	29.60	23.31
bw tu	8	14	8614	2434	28.26	26.72	29.92
se e^{11} tu^{49h}	8	13	8799	3275	37.22	38.11	36.41
tu^{h} (isogenic)	8	13	5464	2421	44.31	42.09	46.86
tu^{g} (isogenic)	8	12	4626	2156	46.61	49.19	43.81
tu^{48j}	8	14	5865	2833	48.30	53.66	43.67
tu^{h}	8	13	12236	6616	54.07	50.98	57.69
vg mt^{A} bw	8	14	10069	5944	59.03	56.49	61.09
y B$^{263-43}$ (homozyg.)	4	7	3120	2274	72.88	69.92	75.77
tu^{g}	8	14	11967	9113	76.15	87.30	65.34
vg bw tu	8	14	10555	10540	99.86	99.77	99.94

Burla, Hans Drosophilids of the Ivory Coast (French West Africa).

During three months, from July to October 1951, I collected Drosophilids at five different places close to Abidjan, Ivory Coast. Two of the places lie in small spots of rain forest along the coast, the third in a secondary forest of the same region, the fourth in a cultivated area with plantations of banana and coffee, and the fifth in a very big mesophile virgin forest 100 km away from the coast. A total of 98 species has been recorded. Only 32 of them occurred on the fruit bait generally used for collecting Drosophila. Thirty species were recorded near the stumps of cut palm trees (Raphia species), 23 species around cut Mahogany trees, 32 species on fungi, 25 on wild fruits, and 5 on flowers. The following genera, subgenera, and groups are represented: Chymomyza (9 species), Leucophenga (9), Zaprionus (9), Hirtodrosophila (3), Mycodrosophila (11), genus Drosophila (44), Pholadonis (21), Sophophora (12), *melanogaster* group (5), subgenus Drosophila (11). The remaining species could not be classified yet. Of the 65 Drosophilid species of the Ethiopian region mentioned by Duda (1939-40), only 22 could be found again, including a few doubtful determinations. About 70 species seem to be new. The characters of many of the species are aberrant and thus lead to revised definitions of the systematical group to which they belong.

Buzzati-Traverso, A. A. Interspecific crossings in the *affinis* subgroup.

Extensive tests have been made to check whether the American species belonging to the *affinis* subgenus (*affinis*, *algonquin*, *athabasca*, *azteca*, *narragansett*) could be crossed with the only known European representative of this subgroup, *D. helvetica*. No hybrids have been obtained.

Buzzati-Traverso, A. A. Interspecific crossings in the *obscura* subgroup.

D. ambigua females, when crossed with *D. miranda*, *pseudoobscura*, or *persimilis* males produce a small number of hybrid larvae. Salivary-gland chromosomes can be studied in the mature larvae. No pairing occurs between the chromosome elements of the two species involved; 13 long and 2 short chromosomes can be seen originating from a common chromocenter. Larvae pupate, but no adult individuals have been obtained.

Buzzati-Traverso, A. A. Interspecific crossings in the *Pholadoris* subgenus.

Crossings between *D. victoria* and *D. lebanonensis* gave both ways perfectly fertile hybrids. Reciprocal crossings between either one of the aforesaid species and *D. nitens* gave sterile adult females. Salivaries have not been studied yet.

Buzzati-Traverso, A. A. Natural selection under increased mutation pressure in *D. melanogaster* populations.

Using the same isogenic strain, four populations in numerical equilibrium were established. While the control population did not receive any treatment, the other three were subjected to 500, 1000, 2000 r units every fifteen days; only adult males were irradiated. From time to time the fertility of females, the egg hatchability, and the total productivity of adult individuals were tested. The experiments showed that an increase in the mutation rate brings about a more rapid rate of adaptation to the environment. The 2000-r population became much more prolific than the control series over a period of thirty generations. The experiments were repeated with different isogenic strains, and confirmed.

Carson, H. L. Interfertile sympatric sibling species within *D. bocainensis* Pavan and da Cunha 1947.

A study has been made of flies collected by Dr. A. R. Cordeiro at a single locality in Rio Grande do Sul, Brazil, all of which were apparently morphologically *D. bocainensis*. According to salivary-gland-chromosome examinations of their offspring, the 51 wild females studied fall into two clear non-interbreeding groups on the basis of the chromosome arrangements that they transmit. Twenty-nine individuals fall into group A, to be designated *D. parabocainensis* n. sp.; these gave offspring homozygous for gene arrangement, except for an infrequent short inversion in 2L. Twenty-two individuals fall into group B, for which the name *D. bocainensis* will be retained. The latter is highly heterozygous for gene arrangements in chromosome 2 and especially chromosome 3. The X chromosome, although homozygous for gene arrangement, could be observed to differ considerably in arrangement from that in group A. Reciprocal crosses between these two in the laboratory, using strains from either within or between localities, produce luxuriant F_1's, in which the hybrid individuals are heterozygous for from 14 to 24 inversions, depending on the strain of *bocainensis* used. There is consistently an 8-inversion difference between the X chromosomes of the two. Except for obvious mechanical difficulties due to the rearrangements, pairing in the hybrid salivary-gland cells is complete, and so far no "small differences" in banding pattern have been detected. The F_1 hybrids of the sympatric or allopatric cross *parabocainensis* female x *bocainensis* male produce a vigorous F_2 and are fertile in all backcrosses; F_1 males from the cross *bocainensis* female x *parabocainensis* male, although they inseminate their sister and backcross females with motile sperm, appear to be largely sterile. F_2's in this direction, however, have been obtained in 4 out of 16 such crosses, but only in mass culture. Three of these four are from sympatric crosses. A third species of the group, to be designated *D. bocainoides* n. sp. has been found in collections from the state of São Paulo. The male of this species is strongly differentiated morphologically from those of the two sibling species, and no species hybrids have been obtained with it. Chromosomal differences also appear to be relatively greater, and the evidence thus indicates that D. bocainoides is quite separate from the other two. Study of natural chromosomal variability, hybridization, and geographical distribution of these three entities is continuing.

Cooper, K. W. The Chapter on Spermatogenesis in Biology of Drosophila. Although the introductory remarks to Biology of Drosophila by Dr. Demerec (p. vi) suggest that Professor Alfred Huettner's material on the spermatogenesis of Drosophila was placed at my disposal for drawing up the chapter on "Normal Spermatogenesis" (and have been so taken by at least one reviewer), in all fairness to both Dr. Huettner and myself let it be said that this was not the case. Regrettably, I have never seen either Dr. Huettner's notes or preparations, nor did I know of their availability until I had read Dr. Demerec's prefactory note in the published volume. The chapter on spermatogenesis was prepared on very short notice, which did not allow extensive original investigation on my part. The slide material used consisted of a large collection of very beautiful slides prepared by Professor Curt Stern, and generously loaned to me, some 40 slides made by myself for elucidation of the first meiotic prophase, and supplementary preparations of living spermatocytes; these formed the basis for such original observations as appear in the review.

de Cunha, A. Brito, Brncić, D. J., and Salzano, F. M. Comparative study of chromosomal polymorphism in populations of tropical Drosophila. Groups of closely related species were chosen for this study: D. grizeolineata and D. guaramunú; D. cardinoides and D. polymorpha; D. bandeirantorum. The results so far obtained are:

Species	No. ind. studied	No. different inversions	Mean no.of inv. heterozygote per individual in different populations
D. grizeolineata	446	5	0,01 - 0,53
D. guaramunú	312	16	1,41 - 2,85
D. cardinoides	80	1	0,01
D. polymorpha	155	6	1,44
D. bandeirantorum	328	2	0,32 - 0,56

The ecological data we have indicate that D. guaramunú is ecologically more versatile than D. grizeolineata, and that D. polymorpha is more versatile than D. cardinoides. D. bandeirantorum seems to be very specialized and common only a short time during the year. The cytological data suggest that the amount of chromosomal polymorphism is proportional to the degree of ecological versatility of the species and to the complexity of the environment where the population lives.

Dale, Ernest E. Differential mortality. An attached-X stock of Drosophila (D. melanogaster) with red-eyed females and white-eyed males gave differential mortality of the two sexes when exposed to culture medium containing colchicine. Data are given on survival of colchicine-exposed flies and controls, the experiments being run simultaneously and covering an eight-day period.

	No. surviving	Per cent
Colchicine exposed		
♀ 700	29	4.1
♂ 700	525	75.0
Controls		
♀ 449	355	79.1
♂ 451	371	82.3

Dresden, D., and Oppenoorth, F. J. Selecting strains resistant to gamma-HCCH (hexachlorocyclohexane).

Starting selection experiments with two strains of D. melanogaster, Berliner Inzucht and a wild strain, an attempt was made to obtain resistance against gamma-HCCH (contact method in adults). The LD50's were 2 and 1 microgram respectively. During 6 selections the LD50's increased gradually to 6 micrograms in both strains. This value did not change during the next 10 selections. By means of a new injection method we will try to find out to what extent the increased LD50 is due to internal or external factors.

Edmondson, M. Interchange of eye and antennal tissue during development.

A Drosophila male was found which had one normal antenna while the other consisted of two joints that were fairly normal except that they lacked an arista and were covered with eye facets of the normal red color. In the anterior edge of the eye on the same side of the head there was a nick, embedded in which and slightly protruding was the bulb of an antenna. It seems clear that there had been an exchange of eye and antennal tissue, each into the place of the other, during imaginal development.

Epling, Carl, and Mitchell, Donald F. A previously unrecorded gene arrangement of D. pseudoobscura in southern California.

The "Pikes Peak" gene arrangement of the third chromosome of D. pseudoobscura is common in the Rocky Mountains, Texas, New Mexico, and Arizona. It has been found in low concentration in central California. It has been unknown in southern California until the present year, although the populations at Keen Camp and Piñon Flat have been sampled repeatedly each year since 1939 by Dobzhansky and, more recently, by ourselves. An approximate total of 14,000 chromosomes has been determined. This year one Pikes Peak chromosome was found in a late-season sample of six females from Piñon Flat. It was also found repeatedly at three other stations in the San Jacinto Mountains not previously sampled. One of these is about six miles from Keen Camp, the others are about four miles from Piñon Flat. Large samples made at these stations throughout the season 1951 indicate that Pikes Peak had a frequency in each population of about 1%. The combinations of Pikes Peak with other gene arrangements present are easily distinguished in salivary-gland smears; thus it is improbable that it has been overlooked in the past even though it is infrequent. The meaning of its appearance, especially at Piñon Flat, is obscure.

Faber, J. and Sobels, F. H. A new imaginal ring in the mid part of the hindgut.

A histological analysis of pupae of the mutant lethal nonevaginated (lne, 1-0.1 ±) revealed the existence of a hitherto unknown imaginal ring in the hindgut, which is responsible for differentiation of part of the rectal region. In lne pupae a delay occurs in the replacement of the posterior part of the larval hindgut by cells from the genital discs. Because of this delayed development it was possible to observe an imaginal structure situated in the mid region of the hindgut developing also independently of the anterior part of the hindgut, which is formed by the imaginal ring caudal to the entrance of the malpighian tubes. These imaginal cells probably give rise to that part of the posterior intestine which represents the rectal ampulla. The same imaginal structure could be observed in normal pupae. Transplantation experiments are under way to check these observations.

Fujii, S., and Kawabe, M. On the development of some "bristle genes" in the pupal stage of D. virilis.

The formation of the bristles in wild flies begins about 48 hours after puparium formation. A study on singed^{14} showed that the formation of bristles is delayed several hours (60 hours after puparium formation), but the time of bristle pigmentation is much the same as that in wild flies (about 100 hours after puparium formation). No difference was found between singed^{16} and wild-type in the formation and pigmentation of bristles.

Gowen, John W., and Stadler, Janice Irradiation effects on viability of D. melanogaster.

Day-old male and female imagoes were exposed to X-rays in 0, 20, 100, 500, 2500, 5000, and 10,000 r doses. Repeated pair matings, representing the 49 combinations, were made. Twenty-six different criteria described the physiological effects. Well-being of irradiated females was measured by total eggs laid, per cent of life devoted to egg laying, and days females lived. Eggs laid decreased linearly with dose until, at 10,000 r, less than fifty eggs or four per cent of control were metabolized and laid. Per cent of life given over to oviposition likewise decreased linearly from 75 to 34 per cent over the range of irradiations. Life spans for the irradiated females were irregularly decreased with higher doses. Duration of life for the males was increased by irradiation of 2500 r or more. Per cent of life during which males were fertile decreased linearly with increasing dosage. Rate of decrease was similar to that for females. Quality of the progeny, as measured by hatchability of the eggs, decreased linearly with increasing irradiation, defective eggs being eight times as great at 10,000 r as with untreated females. Defective eggs, when sperm were irradiated, decreased linearly with dosage of X-rays. The decrease was comparable to that observed for the females. Effects were quantitatively similar for irradiation of eggs and sperm in these data. This observation is contrary to that ordinarily considered true when the measure of X-ray effects is sex-linked lethal mutations in eggs or sperm.

Imaizumi, T., and Kimoto, Y. Cytoplasmic constituents of eggs in early developing stages, and their staining properties.

In developing egg cytoplasm we can recognize yolk granules of larger size and minute granules which disperse through hyaloplasm. These minute granules are 0.5 micron in diameter and globular in shape, and never transform in any stage or medium. Because of their insolubility in acetic acid, they cannot be confused with mitochondria, and are distinguished from ultramicroscopic microsomes by their greater size. They stain with pyronin, toluidine blue, or Sudan III, but not with methyl green or the Feulgen technique. After treatment with ribonuclease or with a cold bath of 10% $HClO_4$, basophilia of the granules mostly diminishes. Contrary to these, the hyaloplasm is acidophile in neutral medium, so it may be surmised that most of the RNA of the cytoplasm is concentrated in these granules.

The staining properties of nuclei and other cytoplasmic constituents are shifted from basophile to acidophile or vice versa according to pH of the medium when stained with buffered double bath of toluidine blue and fuchsin S. The pH values of the shifting zone for each constituent are as follows:

Species	Stages	Nuclei (interkinesis)	Cytoplasm		Yolk granules
			Hyaloplasm	Minute granules	
virilis	preblasteme		4.4	2.4 - 3.0	5.4 - 6.0
	blasteme	4.0 - 5.4	3.4 - 4.4	2.4 - 3.0	5.4 - 6.0
	early blastodermal	3.4 - 5.0 (achromatic part)		2.4 - 3.0	5.4 - 6.0
melanogaster normal	preblasteme		4.4	3.0 - 4.0	5.0 - 5.4
	early blastodermal	4.0 - 5.0 (achromatic part)		3.0 - 3.4	5.0 - 5.4
melanogaster lethal (without X chromosome)	preblasteme		4.4	3.0 - 4.0	5.0 - 5.4

The details will be reported in another paper.

Ives, P. T., and Evans, Alice T. A probable simultaneous double mutation in the Cy sp^2 chromosome.

In DIS-19, page 46, it was reported that a Cy bw sp^2 was recovered from a stock of net b cn bw/Cy sp^2. In DIS-22, page 71, a curious allele, or series of alleles, of the bw of this chromosome was reported, and given the symbol bw^{47j}. In 1949 another allele was found which was bw-like when homozygous, but allelic only to Cy bw sp^2 and not to net b cn bw. On 51f5 we observed that orange (or) of Mossige, DIS-24: 61, is also present in Cy bw sp^2 and that the 1949 bw-like mutant was an allele of Mossige's or and exactly like it in phenotype. At least one of the bw^{47j}-type alleles has proven to be an allele of Cy bw sp^2 or but not of net b cn bw, Mossige's orange, or the 1949 allele. Mossige's or is not present in other Cy chromosomes of our stock list, including Cy pr, Cy sp^2, Cy L^2 sp^2, Cy al^2 Bl lt^2 cn^2 L^4 sp^2, and Cy al^2 lt^2 L^4 sp^2. Although of very similar phenotype, the mutant gene pd is not present in the Cy bw sp^2 or chromosome but cn^2 is. When In(2R)Cy crosses over from Cy cn^2 bw sp^2 or to its homologue and becomes homozygous the result is a bright yellowish-to-orange eye color, darkening with age, and sp^2 wings. The simplest interpretation seems to be that in 45a a double and simultaneous nonlethal mutation occurred at the bw and or loci of the standard Cy sp^2 chromosome. While technically it should be written as Cy cn^2 bw^{45a} sp^2 or^{45a}, it should be satisfactory and much easier to designate it only as Cy bw sp^2 or. The relation of the various bw^{47j}-type mutants of the local population to bw^{45a} and or^{45a} has not been investigated.

Janzer, Wolfgang Studies on cave animal characteristics.

In connection with studies on the evolution of cave animal characteristics, D. melanogaster has been tested. Cultures raised in the dark for 10 generations showed no significant difference as to their photic responses when compared with those raised in the light. Further, stocks with dark body and eye color (se, e^{11}) could be shown to exhibit significantly higher photophilous behavior than those with light body and eye color (w, y Hw). Of 8 different mutants tested (S/Cy, B, w, e^{11}, se, y Hw, ar/ey^D, Berlin-normal), the mutant Bar (B) showed least photophily.

Judd, Burke H., and Lefevre, G. X-ray-induced dominant mutation in *D. melanogaster*.

The frequency of all kinds of visible dominant mutations was determined after irradiation of Canton-S + males and females. In addition the change in incidence of detected mutations after irrodiation was followed by subculturing the experimental bottles at weekly intervals for a minimum of four weeks. Doses of 2500 r and 5000 r were applied in the male irradiations; 3000 r in the female. Dominant mutations are sufficiently numerous to provide quantitative data; over 2% dominants was found as a maximum. However, the number of detected mutants declines rapidly two or three weeks after exposure. In these experiments no leveling off of the mutation rate was observed in the late subcultures, but the incidence of detected mutation was invariably lowest in the last subculture. Moreover, after four weeks the same mutant incidence was observed in both the 2500-r and 5000-r experiments, even though it was much higher in the latter at the beginning. The mutation rate in the 3000-r female series was somewhat, but insignificantly, lower than that in the 2500-r male series. Even in the female irradiations a decline in the incidence of detected mutations was noted after 11 days. Apparently, germinal selection is operative in eliminating induced mutations from the germ line. It cannot be decided if this is the sole cause for the decline, or whether, in addition, an intrinsically lower dominant mutation rate exists in the gonial as compared with the mature germ cells. All the mutants tested were homozygous lethal.

Kikkawa, H. Effects of 3.4-dihydroxykynurenine on pigment formation.

The biochemical step from 3-hydroxykynurenine to brown pigment is quite unknown. But judging from Raper's works for the melanine formation, 3.4- or 3.6-dihydroxykynurenine seems to be a next product of 3-hydroxykynurenine. Then 3.4-dihydroxykynurenine has been synthesized by Drs. T. Sakan and S. Seno of Osaka City University. Tests of this substance to v bw and cn bw mutants of Drosophila were, however, negative.

Koske, Thea A new species hybrid in the *obscura* group.

For some time I tried to cross European species of the *obscura* group, but only with negative results. Later I also included some American species. Some time ago I learned by letter from Professor Buzzati-Traverso that he had succeeded only in crossing *D. ambigua* females with *pseudoobscura* and *persimilis* males of certain origin. (A report has been made recently by him at the Intern. Congress of Entomology at Amsterdam.) Shortly afterwards I obtained a hybrid by crossing *pseudoobscura* females (Oaxaca) with *ambigua* males. The *ambigua* strain was of Swiss origin and highly inbred. The salivary chromosomes of the hybrid show a most complicated pattern. There are some inversions, overlapping inversions, and smaller rearrangements. Some parts lack in pairing. Nevertheless, extended sections of certain elements are exactly paired. An analysis will be carried out. Also the interaction of mutant alleles in the hybrid will be tested.

Lewis, E. B. Additions and corrections to the cytology of rearrangements.

The following salivary-gland-chromosome locations of break points in certain rearrangements supplement descriptions found in the work of Bridges and Brehme.

Additions:

Rearrangement	Break Points
T(1;3) v	10 / 93B
T(1;3)05	4F / 88A-C / 92/62B-C (new order in 3 is: tip of 3L to 62 B-C / 88A-C through centromere of 3 to 62B-C / 92 to 3R tip; section 88A-C to 92 is inserted into X--in 4F according to Griffen's analysis).
T(2;3) A	39 B-C / 83B
T(2;3) B	33 / 81F
T(2;3) 101	42-43 / 83 E-F
T(2;3) 108	The single euchromatic break is in 52D-F and is superimposed on In(2R)Cy.
T(2;3) 109	22F-23AB / 80 / 55F-26A (a cyclical exchange of tips as reported by Bridges and Brehme, but contrary to earlier report the inversion in 3R is evidently In(3R)P.).
T(3;4)c	86B-C (just to right or left of 86C1-2) / 101F
In(3LR) sep (Muller)	65E/85E

Correction:

T(2;3)Xa	The break in 3R which is superimposed on In(3R)P is not in 89D but lies near the end of 89E (to the right of bx and its pseudoalleles).

Lindsley, D. L. An X chromosome specifically deficient for the nucleolus organizing region.

In experiments in which newly derived X chromosomes, involving changes in the heterochromatic region, are recovered, it is desirable to test every product for the presence or absence of each known heterochromatic marker separately. Therefore, a chromosome lacking the nucleolus organizing region but retaining the bb locus and block A has been made. The proximal break of In(1)sc^{L8} is immediately to the right of the nucleolus organizing region, while the proximal break of In(1)w^{m4} is immediately to the left of it (Kaufmann, 1944). A single exchange between these two inversions results in one product which is duplicated for the region from immediately to the right of sc to immediately to the left of w and is deficient for the nucleolus organizing region. This product is viable in heterozygous but not homozygous females. It lives as a male in the presence of Y or Y" but is sterile; XO males or males carrying Y^{1c} do not survive. Such viability data agree with observations that the nucleolus organizing region of the Y chromosome is carried on Y short. The sterility of nucleolusless/Y is puzzling, since males carrying larger deficiencies, also including the nucleolus organizing region, such as In(1)sc^4 sc^8 are fertile; also males carrying duplications for all of the region duplicated in the nucleolusless chromosome and more are fertile (T(1;4) w^{m5L}).

Lüning, K. G. X-ray-induced mutations in different stages of spermatogenesis.

Wild-type, M5, and y w sn males were irradiated (2900 r) at the ages of 0-1 or 6-7 days. The males were mated to virgin y w sn females immediately or after some days. Every day or every third day the males were transferred to new females. Eggs were collected and the number of hatched eggs was counted; total, 150,000 eggs. In the first 5 days the rate of dominant lethals was nearly constant. Then there was a more-or-less sharp increase in the rate of dominant lethals. This high frequency remained till the 11th day; then there was a sharp decrease, which continued to the 20th day after treatment, when there was only a slight effect of treatment compared to the controls. The increase in the rate of dominant lethals appeared at the same time, whether the males were mated or unmated from irradiation to the 5th or the 7th day after treatment. From this it was concluded that when males were unmated the sperm was resorbed and/or spontaneously ejaculated.

To explain the variations in the rate of dominant lethals, it was supposed that the variation fitted with different stages of spermatogenesis that were treated. In the first 5 days, sperm that was mature at treatment was used. In the next 5 days, sperm was used which was maturing (spermatid stage) at treatment. After the 11th day, sperm that was immature at treatment was used.

Later the frequencies of point-mutations (y, w, and sn), gynandromorphs, and hyperploid males were studied in the two periods 1-6 and 7-10 days after treatment. Wild-type males were irradiated (960 r) and mated to virgin y w sn females, two of each sex per vial. At the beginning of the 7th day the males were transferred to another 5 virgin females. The following data were obtained:

Days after treatment	Total females and gyn.	Point-mutations	Gynandromorphs	Hyperploid males
1-6	59856	8	35	12
7-10	47007	22	32	56

From this it is concluded that spermatids are more sensitive to irradiation than mature spermatozoa.

Mainx, F. Structural variety in wild populations of European species of the obscura group.

For some time I and my collaborators have been occupied with this problem. In a short time a paper will appear containing general statements and the salivary chromosome maps of D. subobscura, D. obscuroides, and D. ambigua. A second paper will deal with a qualitative analysis of the inversions in D. subobscura (Austrian, British, and other strains). Further current investigations deal with the selective value of inversions in artificial populations, with quantitative analysis of certain populations, with selection of inversions by inbreeding, and with X-ray-induced inversions in D. subobscura.

Makino, Sajiro, and Kanehisa, Takeharu A preliminary survey of the geographical distribution of Drosophila in Hokkaido.

The species of Drosophila so far collected from Hokkaido and their distribution are preliminarily listed below. For description, the island of Hokkaido was divided into four regions--South, Central, East, and North.

	South	Central	East	North
Amiota sp.		+		
Scaptomyza sp.	+	+		+
Hirtodrosophila sp.		+	+	+
H. cinerea group	+			
melanogaster		+		
auraria	+	+	+	+
suzukii	+	+	+	+
obscura group		+	+	
transversa	+	+	+	+
nigromaculata	+	+	+	+

	South	Central	East	North
virilis	+	+	+	+
testacea	+	+	+	+
funebris	+	+	+	+
histrio		+	+	
immigrans		+	+	
rufa		+	+	
repleta group			+	
melanica group			+	
macularis group			+	
robusta group	+	+		
sordidula		+	+	+
coracina		+	+	
busckii	+	+	+	
Mycodrosophila sp.			+	
grandis (?)			+	

Makino, Sajiro, and Kanehisa, Takeharu A monthly survey of Drosophila in the City of Sapporo, Hokkaido. Monthly collections of Drosophila have been made in three different regions in the City of Sapporo. The species so far observed and their monthly appearance are as follows: (V = May, VI = June, VII = July, VIII = August, IX = September)

	West					South					North					Total
	V	VI	VII	VIII	IX	V	VI	VII	VIII	IX	V	VI	VII	VIII	IX	
auraria	145	300	426	315	109	-	41	246	437	27	-	16	50	91	18	2221
nigromaculata	115	80	229	196	-	-	-	195	44	-	-	-	12	5	-	866
transversa	-	-	16	19	5	-	-	28	6	21	-	-	-	-	2	97
immigrans	-	-	64	111	51	-	-	16	15	3	-	-	11	3	1	275
funebris	-	-	13	-	5	-	-	26	10	3	-	-	5	-	-	62
busckii	-	-	-	63	-	-	25	24	68	-	-	-	-	-	-	180
testacea	-	20	25	5	4	-	-	59	12	-	-	-	-	-	-	125
histrio	-	-	6	-	-	-	-	-	5	-	-	-	-	-	-	11
coracina	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	1
melanogaster	-	-	-	34	15	-	-	-	-	2	-	-	-	-	-	51
virilis	-	-	-	9	2	-	-	-	33	2	-	-	-	-	-	46
robusta gr.	-	-	-	51	-	-	-	-	4	3	-	-	-	-	-	58
rufa	-	-	1	-	-	-	-	-	-	-	-	-	-	-	-	1
Scaptomyza sp.	-	5	-	-	-	-	-	9	-	-	-	-	-	-	-	14
Total	260	405	780	803	191	-	66	603	635	61	-	16	78	99	21	4008

Makino, Sajiro, and Takada, Haruo A monthly survey of Drosophila in the vicinity of Otaru City, Hokkaido. Monthly collections of Drosophila have been made in Otaru City and its vicinity, Shioya, in Hokkaido. The species so far observed and their monthly appearance are listed below: (V = May, VI = June, VII = July, VIII = August, IX = September)

	Otaru City					Shioya					Total
	V	VI	VII	VIII	IX	V	VI	VII	VIII	IX	
auraria	+	+	+	+	+	+	+	+	+	+	394
nigromaculata	+	+	+	+			+	+	+		177
transversa	+	+	+	+	+		+	+	+	+	
sordidula				+			+	+	+		9
immigrans			+	+	+				+	+	120
funebris				+	+						5
repleta					+					+	8
melanogaster				+	+					+	206
virilis				+	+					+	7
testacea					+					+	4
suzukii					+					+	30
buskii					+					+	11
histrio										+	1
robusta group										+	3
Amiota sp.										+	2
Total	27	53	89	120	197	38	71	88	102	192	977

Makino, Sajiro, and Kanehisa, Takeharu Some notes on a heritable tumor found in D. virilis.

Individuals having a tumor-like outgrowth in the head and thorax regions were found in outbreeding stocks of the wild strain of D. virilis collected in Sapporo, Hokkaido. It occurred in 53 specimens out of 1224 individuals under observation. In general, the tumor-like outgrowth was observed to develop especially in individuals showing abnormalities in wings and others. Results of inbreeding with the tumor-bearing flies are listed below. The frequency of tumor-bearing individuals tends to increase with the generation of inbreeding. (Temperature: 20°-27° C.)

	Indv. with tumor		Tumor indv. with bodily abnor.		Normal indv.		Indv. with bodily abnormalities		% of tumor indv.
	♀	♂	♀	♂	♀	♂	♀	♂	
F_1	37	32	41	26	48	74	27	22	43.8
F_2	40	27	4	10	22	24	7	8	58.0
F_3	37	10	8	9	4	0	0	2	91.0
F_4	202	132	16	22	17	30	3	13	92.0

Meyer, Helen U. Evidence of the unsplit condition of interphase chromosomes.

A Notch mutant had been induced in a pole cell of a male embryo by treatment with ultraviolet (irradiated in the polar cap stage in a nitrogen atmosphere, in which, however, it was kept for only 7 minutes). Breeding analysis showed that in addition to this lethal in the X chromosome, lethals had also been induced in both second chromosomes of the same pole cell. This cell proved to be the only primordial germ cell which was furnishing functional sperm, as evident from the fact that all female-producing sperm derived from this male transmitted the Notch in the X chromosome and all sperm analyzed transmitted one or the other of the two second-chromosome lethals (depending on which homologue of the second chromosome had been received, as shown by markers present).

This case allows the following conclusions to be drawn: (1) Since

Notch is lethal to males, the male which grew up from the treated embryo could not have received the Notch mutation from its mother; in all probability, then, it was induced by the treatment. This gives strong evidence that, when pole cells are treated, not all cases where the same lethal is found in all gametes receiving a given chromosome have been caused by the pre-existence of the lethal in one of the parental germ cells; they must sometimes be caused by induction of the lethal in one pole cell while the other pole cells are killed off. (2) This particular Notch is seen not to have acted as a cell lethal during the stages of spermatogenesis, even though the X chromosome was present in haploid dose, and all the germ cells had Notch in their X chromosome. (3) Though only one functional primordial germ cell was left, about 50 female offspring were obtained, and about the same number of male offspring (of which 43 were analyzed for mutations in their second chromosomes).

Now since all offspring receiving a given treated chromosome showed the given induced mutation (whether Notch or autosomal lethal) it could be concluded--as pointed out by H. J. Müller--that in all probability these mutations must have been present in both chromatids that were derived from the given treated chromosomes of the survivor pole cell. This indicates strongly that none of these chromosomes were split into two daughter chromatids during interphase, since cells of the polar cap are known to be in interphase at the time chosen for treatment. This conclusion could be avoided only by supposing exactly the same mutation to have been simultaneously induced in both chromatids in the case of all three chromosomes--an assumption which seems highly unlikely--or by supposing that one of the two daughter cells of the surviving pole cell failed to give rise to any functional germ cells. The last supposition also appears unlikely. This because, firstly, the pole cells do not undergo division until some 14 hours after the stage at which they are treated. Therefore a physiological injury would be unlikely to have remained so localized until that time as to kill off one daughter cell completely while allowing the other one to give rise to numerous descendant germ cells. Secondly, the cells are by the time of this first post-polar division located within the definitive gonads, but for some divisions after that still appear to be entirely undifferentiated from one another. Thus it also seems very unlikely that one of the two daughter cells produced by a given pole cell at that stage would, through its later divisions, give rise to a multitude of functional germ cells, while the other daughter cell gave rise exclusively to nongerminal cells. Conceivably, however, one of the two daughter cells might have been killed by an unrestituted break or other dominant lethal genetic change that had occurred in only one of a pair of sister chromatids.

During our 4 years of work on the induction of mutations by ultraviolet treatment of the polar cap, three other cases had been found (but none in the about equally numerous controls) in which all tested offspring that had received a given second chromosome contained an identical lethal, while at the same time all the other offspring, which received the homologous second chromosome, contained another lethal, identical in all the latter. These cases either resulted from coincidence of pre-existing lethals in both the paternal and the maternal second chromosome or were due, like the case of Notch reported above, to one surviving germ cell with lethals induced in both second chromosomes, which also were presumably in an unsplit condition during interphase, in which stage the pole cells are known to be.

Mickey, George H. Origin of a new R allele in mosaic D. melanogaster.

A new dominant allele of Roughened, which is lethal in homozygous condition and which produces an extreme eye surface resembling glass or Glued when heterozygous with

Roughened, arose in a single mosaic male among 102 progeny of a female treated with cold shock at an early embryonic stage. The mosaic male produced 788 offspring, only one of which showed the roughened eye. The latter produced 50% offspring, rough in both sexes, indicating its normal viability in a heterozygote. Consequently it appears that possibly a single cell carrying the mutant was incorporated into the mosaic gonad of the original male. This observation is significant in considering mutation rates resulting from treatment of pole cells.

Mickey, George H., and Blount, Jerry Somatic polyploidy in *D. melanogaster* induced by cold shock.

The effects upon somatic cells (ganglia) of cold shock applied to embryos at the pole-cell stage and to third-instar larvae of *D. melanogaster* were investigated. Results were scored from aceto-carmine squash preparations of the third-instar larval brains by comparing the ratio of normal diploid metaphases to the polyploid metaphase figures. Results were as follows:

Stage	Treatment	No. Indiv.	No. Figures	No. Poly.	% Poly.
control	untreated	53	1746	0	0
pole cell	1/2 hr. -5.5° C	22	957	38	3.3
pole cell	2 hr. -3.3° C	19	976	102	9.1
pole cell	1 hr. -6.1° C	16	729	106	12.0
third instar	24 hrs. -6.1° C recover 24 hrs.	9	763	289	29.7

Both temperature and length of treatment influence polyploidization but the temperature appears to be relatively more important. The last experiment gave the highest percentage of tetraploid cells and the most consistent figures. This may be due to the fact that there was less opportunity for the elimination of these tetraploid cells before their detection. Gloor's treatment of *D. hydei* larvae at higher temperatures (8°-12° C) and for a longer period (10 days) gave a much higher degree of ploidy (Gloor, DIS-24: 82). The length of treatment in our experiments allowed for only a single doubling of chromosomes.

Mickey, George H., and Di Paolo, J. A. Lethals induced in Drosophila by combined action of urethane and H_2O_2.

Adult males of *D. melanogaster* were injected with M/4 urethane (ethyl carbamate) in Holtfreter's salt solution. From 12 males a total of 641 chromosomes were tested for sex-linked lethals, using the Muller-5 technique. Only 2 lethals were detected (from separate flies). From 23 males injected with the same solution of urethane but also treated for 24 hours with fumes of superoxol (3% H_2O_2) a total of 1203 chromosomes were tested and 17 lethals were detected. Two flies gave three lethals each, three gave 2 lethals each, and the others only 1. Crossover tests proved all the cases of multi-lethals from single males to be distinct (with one possible exception). The rate of lethal production in the experiment using uerthane (0.31 ± 0.22%) was not significantly different from the controls (0.26 ± 0.12%); but the percentage of lethals induced by the combined treatment (1.41 ± 0.34) was significantly greater than in either the controls or the urethane experiment.

Mickey, George H., and Sturtevant, F. M., Jr. Failure of phenol to produce lethals in Drosophila.

Phenol had been administered to Drosophila previously by subjecting adults to vapors, injecting adults, soaking fertilized eggs or excised ovaries, and placing phenol in the food. We employed three methods hitherto unused for phenol: (1) male third-instar larvae and adults of both sexes were subjected to a constant flow of phenol vapors for 24 hours; (2) different concentrations of phenol (0.20%, 0.25%, and 0.50%) in Holtfreter's saline solution were injected into third-instar male larvae, and 0.50% into female larvae, using a semi-micropipette; and (3) mature sperm were treated with phenol (0.01, 0.1, 1.0 and 2.0%) in Holtfreter's solution by the vaginal-douche method of Herskowitz. Control and experimental series were tested for sex-linked recessive lethals by the Muller-5 method. Rate of lethal production in experimentals was in no case significantly different from that in the controls. The reason for the failure of *in vivo* treatment is postulated to be phenol detoxication in the fly and inability of the phenol to reach the germ plasm during the critical physiological period.

Miller, D. D. Mating behavior in *D. athabasca* and *D. narragansett*.

Observations of mating behavior in *D. athabasca* and *D. narragansett* are in progress, employing New York and New Jersey strains of *athabasca* (kindly supplied by Drs. E. Mayr, C. Pittendrigh, and B. Wallace) and a New Jersey strain of *narragansett* (furnished by Dr. C. Pittendrigh). A number of differences between the mating behaviors of these species have been observed, both with respect to each other and with regard to the similar species *D. affinis* and *D. algonquin* (Miller, 1950). *D. athabasca* males were found to be different from males of the other three species in regularly extending and vibrating one wing rather than both during courtship. A distinctive courtship movement of *D. narragansett* males was rapid opening and closing of the wings while approaching and circling about the females. The following table presents data on copulation times in the four *affinis* subgroup species *affinis*, *algonquin*, *athabasca*, and *narragansett*.

Temp.	*affinis*	*algonquin*	*athabasca*	*narragansett*
27° C			1'13", 1'26", 1'12", 1'23"	
26			1'25"	
25				10'59", 14'32"
24	58", 1'10", 1'13", 55", 1'1", 1'26"	5'35", 4'50", 7'31"	1'40"	
23	1'24", 1'3", 1'29", 1'24", 1'22"	4'37", 5'42"	1'48", 1'42", 1'27", 1'15", 1'12", 1'25", 1'25"	17'42", 20'52"

A few interspecific mixtures of males and females have been observed. Attempted (but not successful) copulations have been observed in both reciprocal combinations of the species pairs *algonquin* x *athabasca* and *athabasca* x *narragansett*.

Mittler, S. Variation of the penetrance of tu^{50j} when reared on yeasts that do not require vitamins or amino acids.

A highly inbred stock of *D. melanogaster* containing tu^{50j} was raised on a minimal medium consisting of glucose, $(NH_4)_2SO_4$, and several trace elements, inoculated with yeasts that were able to live in absence of vitamins or amino acids. Hence, the flies obtained practically all their nourishment from the yeast and not from the medium. In research work involving penetrance and expressivity, utmost attention is usually given to temperature, and the nutritional aspect is almost always ignored. The following yeasts were used and are presented in a series decreasing in the ability to aid in the formation of tumors: *Hansenula anomala*, *Pichia membranaefaciens*, *Candida sorbosa*, *Nadsonia fulvescens*, *Debaromyces globosus*, *Hansenula saturnus*, *Torulopsis utilis*, *Rhodotoryla gracilis*, *R. glutinis*, and *Geotrichium*. Penetrance was less when the above yeasts were used compared to *Saccharomyces cerevisiae* (Baker's yeast) on cornmeal-molasses medium. *D. melanogaster* can live exclusively on a nonfermenter yeast, *Pichia membranefaciens*.

Moriwaki, D., Okada, T., Ohba, S., and Kurokawa, H. Drosophila species belonging to the "*obscura*" group found in Japan.

In the summer of 1951, we were able to collect about 800 flies (females about 160), belonging to the "*obscura*" group of Drosophila, at several localities in Hokkaido (Akkeshi and five others) and one locality in the northern district of Honshu (Mt. Hakkoda). Although it still remains undecided whether these flies form one species or more, they are believed to belong to the "*obscura*" rather than to the "*affinis*" subgroup.

Having compared them with ten species of the "*obscura*" subgroup, namely, *pseudoobscura*, *persimilis*, *miranda*, *obscura*, *subobscura*, *obscuroides*, *tristis*, *bifasciata*, *alpina*, and *ambigua*, mostly according to descriptions seen in the literature, we found that the several characteristics, such as color of mesonotum, male sex-combs, male genitalia, and karyotype, of this species, if it is one, were most similar to the descriptions of *D. obscura* Fallen.

On the other hand, the "*obscura*" species of Sweden described by Fallén (1823) may be identified as "*subobscura*," as proposed by Buzzati-Traverso (1949) on the evidence that "*D. subobscura* is numerically prevalent at Esperöd (Sweden) and has the wider geographical distribution in continental and insular Europe among species of the *obscura* group." The "*obscura*" species of Moscow described by Frolova & Astaurov (1930) has a karyotype of either "A" (♀: V-shape 4, Dot 1) or "B" (♀: V-shape 3, Rod 2, Dot 1), either of which differs from the karyotype of *D. subobscura* showing Rod 5 and Dot 1. Then the Swedish *obscura*, provided that it should be considered as being *subobscura*, seems to be different from the Moscow *obscura*. Moreover, the karyotype of the present species in Japan coincides with the "A" type, one of the two types of the Moscow *obscura*.

At any rate, *D. obscura* is an uncertain species, as pointed out by Buzzati-Traverso in DIS-23 ("What is *Drosophila obscura*?"), and the identification is very difficult. But it is desirable to decide early to which species the name "*obscura*" should be given, in order to establish the synonymization.

Mossige, Jeanne Two new jaunty mutations.

This laboratory has had one stock containing j, namely, b j pr cn. On Oct. 18, 1949, one sv^2 male was found in sv^2 stock with curled wings. This proved to be an allele of j. On May 5, 1950, several sc cv v f flies in sc cv v f stock were also found to have curly wings and these too were j. The occurrence of two new spontaneous j mutations in the same laboratory within such a short space of time seems remarkable, as only two alleles have been reported previously. Contamination would seem to be impossible as the stocks where the mutations were found showed no irregularities and if contamination had come from b j pr cn then the other markers should also have been found. Moreover the first mutation has been kept in combination with sv^2, which again should have been found in sc cv v f if the first had contaminated the second. The two alleles, j^{49j} and j^{50e} (see New Mutants) seem to be identical: both have a slighter manifestation than j, both overlap + at 21° but not at 30°, whereas j does not overlap + at 21°, tested at the same time as the others.

Muller, H. J. Detection of mutations in the second chromosome by use of the "sifter" stock.

Flies one or more of whose second chromosomes are to be tested for the presence of recessive mutant genes are first crossed with a stock (such as Indiana stock g98) containing S^2 and Cy in the same chromosome. If the usual inversions in both right and left arms are present with Cy, and preferably also Bl and L^4 as a check on the rare crossing over which these allow, F_1 females as well as F_1 males are available for the testing; otherwise only F_1 males are used. The F_1 flies are crossed individually to flies of the "sifter" stock (Indiana stock j42). In this stock, one second chromosome, containing S and Sp in the left arm and P^- (Pale deficiency) in the right arm, has its right arm connected by a translocation with a third chromosome having the complex of inversions designated as InsCXF, which effectually prevent crossing over with the other third chromosome. The other second chromosome contains Cy, with its left- and right-arm inversions, as well as cn^2, L^4, and sp^2; and the other third chromosome contains the closely linked variants Dl and H and, very near to them, P^i (the Pale insertion, complementary to P^-) and e. Thus the cross of the F_1 flies by sifter flies is as follows (representing by mu the chromosome in which the presence of mutant genes is to be determined, and allowing the presence of the Cy inversions to be understood).

$$(F_1)\ \frac{mu}{S^2\ Cy\ Bl\ L^4\ sp^2}\qquad X\qquad (\text{sifter})\ \frac{S\ Sp\ T23\ P^-\quad .\ InsCXF}{Cy\ cn^2\ L^4\ sp^2\ ;\ Dl\ H\ e\ P^i}$$

If we neglect crossovers, we find that the only F_2 which survive are those having the composition $\frac{mu}{Cy\ cn^2\ L^4\ sp^2}\ \frac{\quad}{Dl\ H\ P^i}$. All zygotes which received one of the T23 chromosomes from the sifter parent will of course die unless they received the other one also, thus getting S Sp T23 P^-. InsCXF. But in that case they fail to receive P^1 and hence are killed by their P^-. Zygotes which received the Cy cn^2 L^4 sp^2 and Dl H P^i chromosomes from their sifter parent can live only if they receive mu from the F_1 fly, for otherwise they will be homozygous for Cy. (Very seldom Cy homozygotes live; in the cross shown they would be recognizable by having Bl).

If the sifter parent was a female there will be a not negligible amount of crossing over between the chromosome arms containing the Cy inversions, because of the reduction of crossings over in the third chromosomes occasioned by InsCXF. The crossovers containing P^- will still die, as do the noncrossovers with P^-, but the crossover gametes of type S Sp cn^2 L^4 sp^2; Dl H P^i will be able to live provided they become combined with the mu-containing gametes of the F_1 (those combined with the S Cy Bl L^4 sp^2 gametes are killed by their S^2/S compound condition). These surviving crossovers would be detrimental to the mutation study if the females were allowed to breed, but they are recognizable by reason of being non-Curly. Hence the flies of F_2 must be etherized and the non-Curlys discarded. Although some of the Curly females may have been inseminated by crossover non-Curly males, this is not a source of error for the recognition of lethal and other mutations in F3, since even the crossover males carried a noncrossover mu chromosome, distinguishable from its homologue through the presence of S and Sp in the latter.

The procedure therefore is simply to mate together, en masse, the Curly (non-Bl) F_2 flies of each culture, and then to examine the F_2 to see whether non-Curly non-Lobe flies are present (their absence indicating a lethal), or are reduced in number relative to the Curlys (an indication of a detrimental), or are sterile (because of a recessive sterile), or visibly abnormal. If evidence of the presence of a crossover chromosome is found, as shown by S and Sp, flies of this kind are eliminated and F_3 virgins without these markers are chosen to continue the line. Thus the F_3 provide the basis for readily establishing a stock, not requiring further selection, of any second chromosome mutant found.

The chief advantage of the "sifter" method is, as its name is intended to imply, its almost automatic sifting of the F_2 zygotes, so as to make it unnecessary to obtain virgins from the numerous F_1-F_2 cultures, at the very stage of operations where this requirement has usually constituted the limiting factor on the size of the body of data obtainable. As abundance of data is of especial importance in most mutation work, the door is thereby opened to studies which would not hitherto have been feasible. The method is applicable to the study of second chromosomes of virtually any composition, and therefore can be used not only for determinations of the frequency of occurrence of mutations in the laboratory but also for the analysis of the composition, as regards their second chromosomes, of flies taken from populations existing in nature.

The warning must however be given that sifter flies are rather weak and not good breeders, especially in view of the few viable offspring they produce on crossing. Therefore several virgin sifter females should be provided for every F_1 male bred, and vial cultures provided with the usual anti-mold compounds and with sterilized cellucotton and cotton. Under these conditions ample F_2 are usually forthcoming for the mass cultures of the next life cycle, the critical cycle; and the next generation develops much better.

Muller, H. J. Homosexual copulation in the male of Drosophila, and the problem of the fate of sperm of males isolated from females.

In a culture vial containing about 20 wild-type males which had been kept (at room temperature) isolated from females, ever since their hatching about two weeks previously, a male was observed to be in copulation with another male. The copulating male was dorsad to the other, in approximately the normal position for Drosophila copulation by a male, except that its abdomen was inflexed farther forward, with the extruded penis firmly fixed into the deep space of the neck region on the dorsal side of the other fly, between the posterior surface of the latter's head and the anterior surface of its thorax. The male being copulated with went about as if undisturbed. The copulation lasted about five minutes from the time it was first observed, and so it may have lasted for the normal length of time.

The question arises, to what extent is this a usual method whereby Drosophila males kept apart from females but in company with other males lose their sperm. The work of Harris (1929) showed that the sperm of males kept without females for some two weeks after irradiation still had the high mutation frequency characteristic of sperm irradiated when mature, instead of the low frequency characteristic of sperm derived from similarly irradiated males after they had been kept with females for about two weeks. However, later work has shown that after not much longer than two weeks following irradiation the males that have been kept without females do show a drop in mutation frequency. If, as seems likely, this drop is not entirely due to the admixture of newly formed sperm with the old sperm, the old sperm must after a time become unavailable. There are several conceivable ways in which this could occur: (1) the old sperm when kept in a male might after a time degenerate, possibly becoming resorbed; (2) the sperm might gradually leak out; as in some vertebrates, they might be lost (3) by ejaculations occurring in the absence of copulation, or (4) in the course of copulations other than heterosexual ones. We now see that possibility (4) does occur sometimes in Drosophila, as in vertebrates, in the form of homosexual copulation, but there is as yet no evidence as to how common it is, aside from the negative fact that it has not hitherto been reported.

Muller, H. J. Localization of Y:bw$^+$ insertion and cr-u sterile (crs). The portion of the right arm of the third chromosome inserted into the long arm of the Y in the Y:bw$^+$ chromosome found by Dempster is very similar in length and position to the Pale insertion (P^i) and can be substituted for it for saving the life of individuals having the Pale deficiency (P$^-$) in one second chromosome. However, it does not extend quite so far to the right as does P^i, so that a Minute bristle effect is produced, by a Minute locus lying between the right breaks of these two insertions. Also between these breaks is the locus of the recessive male-sterility gene, "crs" (called "cru-sterile"), originally associated with "cr-u." The symbol "cr-u" represents Bridges' "cream-underscored," a dilutor of eosin, which he had localized in the left arm of chromosome 2 and which he had thought to be itself responsible by a pleiotropic action for the male-sterility effect. Thus males homozygous for crs are rendered fertile if they contain P^i in one of their third chromosomes, but not if they have Y:bw$^+$ (and no P^i).

Muller, H. J., and associates. Ultraviolet induction of mutants at loci at which spontaneous mutants are known. The question has been raised (by Dr. J. Schultz in a personal communication) whether any mutations which arose after ultraviolet treatment have been found (or made very probable) to be allelic with known spontaneous mutants. The following is a list of such cases known to our group.

X Chromosome

achaete: 1 case by McQuate in 1949, in sc.Y^1 chromosome, not associated with mutation to y or lethal.

fused: 1 case by Edmondson, 1951.

Notch: 1 case by Altenburg about 1930; 1 case by Muller about 1941; 1 case by Meyer, 1947; 1 case by Meyer and Byers, 1951.

vermilion: 1 case by Edmondson, 1951.

Chromosome 2

apterous: 1 case by Meyer and Byers, 1949.
black: 2 cases by Meyer, 1950, 1951.
dachsous: 1 case by Meyer, 1951.
giant larvae: 1 case by Meyer, 1950.
light (lethal): 2 cases by Meyer, 1950, 1951.
Lobe: 1 case by Edmondson and Meyer, 1949.
straw: 2 cases by Meyer, both in 1951.
lethals: various cases of allelism of uv-induced with spontaneous lethals

Probable alleles where the locus has not been tested:

X Chromosome

narrow abdomen: 1 case by Edmondson, 1951.

Chromosome 2

Dent: 3 cases, 1 dominant and 1 recessive by Meyer, and 1 dominant by

Edmondson, in 1950 and 1951. These were all shown to be in the left arm of chromosome 2.

Known alleles which were probably induced, but may be spontaneous:

Chromosome 2

dumpyTh: 1 case by Meyer, 1951.
dumpy-oblique: 1 case by Meyer and Byers, 1951.

Chromosome 3

claret: 1 case by Meyer, 1950, sterile in homozygous female.
glasslike: 1 case by Meyer, 1949. This is an allele of a spontaneous mutation like glass, in the third chromosome, found in this laboratory, but not tested for allelism with glass itself, to which both may be allelic.

Nakamura, K., Inmaizumi, T., and Kitazume, Y. Amino acids in D. melanogaster. Surveys were made by two-dimensional paper chromatography of the free amino acids found in alcoholic extracts of larvae, pupae, and adults, respectively. In each stage, 17 kinds of amino acids were found; leucine, phenylalanine, valine, proline, tyrosine, arginine, histidine, alanine, lysine, threonine, glycine, serine, asparagine, glucosamine, glutamic acid, aspartic acid, and cystine, besides two unknown ones. Of these, leucine and cystine were present in greater quantities in larvae than in pupae and in adults; smaller amounts of phenylalanine were found in adults than in larvae and in pupae. Hydrolysates of normal, lethal (YY), and unfertilized eggs were tested by two-dimensional paper chromatography. Leucine, phenylalanine, valine, proline, tyrosine, alanine, arginine, histidine, lysine, threonine, glycine, serine, aspartic acid, and two kinds of unknown elements were found in each of them. A third unidentified one (cystine?) was found in lethal and unfertilized eggs, but was lacking in normal eggs.

Nolte, D. J. Secondary genic products. A long-term investigation has been undertaken on the eye-pigmentary system of Drosophila, with particular reference to the eye-color mutants of D. melanogaster, the main techniques being a histological study of eye structure and a spectophotometrical assessment of the pigments. Part of the work has been published, several papers are in the press, and further work is in progress. The mutants include 30 of the main eye-color genes, 24 multiple alleles of ten of the foregoing genes, and 4 position effects; 3 wild-type strains are being used for comparison, one being a South African strain.

Four regions of pigment concentration have been located in the compound eye: the primary, secondary, basal, and post-retinal; great variation occurs in the various mutants with regard to the arrangement of the cells, their size, the size of the pigment granules, and the type of pigment contained. The content of brown pigment varies independently of the content of red pigment in the series of mutants already tested, and the color of the eye is not directly proportional to the amounts of the two pigments, but often dependent on the ratio between these amounts. In two series of multiple alleles already tested, one shows a simple quantitative proportional ratio between the two pigments, but the other shows more of a qualitative ratio or relationship, in that the two pigments do not follow the same series of increases in quantity. Although in general the two pigments of any specific strain seem to vary independently in quantity from culture to culture, there appears to be some connection between them at one or another stage of their synthesis; there appears to be, in some mutants, a competition for an assumed common substrate, and thus it was found that pr has more brown pigment than the wild-type, while cd has more red pigment.

As the work progresses it is becoming more and more evident that many of these eye-color genes are such only by nature of some secondary reaction--for example, the provision of a by-product of their primary action, this by-product being utilized in the synthesis of one or the other, or both, of the pigments. While some genes, as for example w, v, cn, bw, might be assumed to be primary eye-color genes, others, as for example rb, cm, g, car, do not appear to be connected with a series of basic stages in pigmentation. In compounds between these genes no epistasis appears, but instead a sub-additive type of interaction, and sometimes even a super-additive interaction as in the case of the compound rb g car, which produces more red pigment than the compound rb car.

Novitski, E. Autonomy of sterility of transformed females.

Using the unstable ring $In(1)X^{c2}$, w^{VC}, which has been shown by Griffen and Lindsley to give rise to a high frequency of gynandromorphs, it was possible, from transformed females of the constitution $In(1)X^{c2}$ w^{VC}/In(1)dl-49, y Hw m^2 g^4 f^5/Y; tra/tra, to select mosaics in which the ring had been eliminated in early cleavage, giving rise to part normal-male and part transformed-female tissue. Of 24 such mosaics, 16 were sterile and the remaining 8 produced, among their 1595 female offspring, no progeny carrying the ring-X which would have been indicative of the normal proliferation of 2X+Y spermatogonia in the mosaics.

Novitski, E. The compound X chromosomes.

There are four ways in which two single X chromosomes may be arranged to give simple compounds since (1) the order of loci may be mirror-image or tandem, and (2) the centromere may be median or terminal. Since each type is unique in its pairing configuration and gives different kinds of information about crossing over, the writer wishes to suggest that each type be given a simple designation. Although the type that is generally called attached-X had, in the earlier days, been referred to at times as double-X, and the double-X of Muller has, more recently, been described as an attached-X, it is felt that a simple consistent scheme is provided by referring to those compounds with median centromeres as attached-X's and those with terminal centromeres as double-X's. In the ordinary attached-X, as well as in Muller's double-X, the arrangement of the two chromosomes is in mirror-image fashion. Consequently, the distinction between the mirror-image tandem arrangements may be adequately provided by referring to the latter as tandem double-X's, as the case may be, and by using no further designation for the mirror-image types.

Two new types have been recently derived; these are (1) the double-X and (2) the tandem double-X.

(1) The double X. Pairs by simply folding back on itself (like the attached-X). Unfortunately, from the standpoint of further genetic analysis, the double-X discovered by Muller and now commonly used instead of the attached-X for stock keeping carries In49 in the heterozygous state. Two new cases have been discovered in the progeny of sc^8 f v cv/y w females where, apparently, a crossover occurred between the distal heterochromatic region of sc^8 and the base of the normal chromosome. The double-X is designated structurally as Normal X + sc^8 and genetically as y w + f (?) v cv sc^8. Homozygosis for the heterozygous mutants may be achieved by double crossing over (singles produce bridges in this type of compound X), and both cv and v appear with a low frequency. Cases of homozygosis for forked have not been

found. There is some doubt, therefore, that this double-X carries that gene. Examination of ganglia show that the chromosome is very long at anaphase (and therefore is of the double type); at metaphase, the chromosome appears V-shaped, but a tip of the V rather than the apex is directed toward the center of the plate.

(2) The tandem double-X. Pairs by forming a spiral and, by crossing over, manufactures single rod chromosomes. This compound was derived by inducing a crossover between the short arm of the Y at the tip of the X·Y chromosome and the base of sc^4. Structurally, the chromosome may be written as sc^4 + EN· Y^{Lsc8}; the raised dot indicates the position of the centromere. The sc^4 chromosome used carried y car m w^a; the X·Y chromosome may be represented as car^+ m^+ y·y^+. Crossover studies indicate that a striking excess of single chromosomes is recovered; it is presumed that crossing over is occurring with normal frequency but that nonrandom disjunction favors the inclusion of the single X in the egg nucleus. The long arm of the Y, present at the base, has been replaced by the duplication B^S. Such a chromosome should, with some low frequency, yield instances where crossing over has occurred between this duplicating fragment at the base, and the homologous region at the end of the sc^4 chromosome. This chromosome would be a double ring, of interest because the recovery of single rings from it by crossing over would have to depend on certain three-dimensional properties of this type of chromosome.

Novitski, E. Useful derivatives of the X·Y chromosome.

Among a large number of rearrangements involving the X·Y chromosome found while looking for a special type of inversion, two may be of particular interest in experiments where the inverted sequence of the X·Y chromosome is troublesome because it gives either too much, or too little, crossing over when heterozygous with a normal sequence. One of these, labeled X·Y In X·Y 26, has one break in the heterochromatin, the other in section 10A. No crossovers have been found when this chromosome is with sc^8 or a normal chromosome. Another, X·Y In X·Y 24, appeared to be an almost complete reinversion since it crosses over freely with a normal sequence. The crossover class with the left end of a normal chromosome and the base of In X·Y 24 is inviable in the male, however, indicating that some normally proximal genes, located distally in the X·Y chromosome, had not been shifted back into their normal position by the second inversion. To make a chromosome that would be free of this complication, In X·Y 24 was crossed to an attached-X detachment (A2) which carried the long arm of the Y chromosome. A single crossover replaced the deficient base by a normal base. The chromosome thus constituted has been tested by mating to y^2 su^{wa} w^a bb/0 females. F_1 males are fully fertile and viable, although there must still be a small duplication at the distal end. It is carried in stock as: Ins 24L + A2R y/y^2 su^{wa} w^a bb and Ins 24L + A2R y v/y^2 su^{wa} w^a bb.

Oftedal, Per Genetics and histogenesis of a new tumor, tu(2)49k.

A tumor stock developed spontaneously from a stock of ma^{49d} shows black aggregations in about 50%-60% of the flies in stock. The tumor may be located in any part of the fly, but usually in the abdomen. In outcrosses 2% incidence is obtained in F_2. In backcrosses the incidence is up to 10% and may rise to 34% when crossed to the stock from which it arose. The main gene is completely recessive and is located in the second chromosome, probably between c and px. It is not an allele of mt^A of Hartung (Hartung, J. Hered. 41: 269). Modifiers have been located in the middle of chromosome 3, rather closely linked to ma, and in chromosome 1. The third-chromosome modifier is present in the Sb and H chromosomes of the Cy/Pm; H/Sb stock in this laboratory.

Histologically the tumors start as a melanization of the larger lymphocytes, visible at 34 hours after hatching, and so complete as to sometimes obscure the presence or absence of a nucleus at 75-78 hours of age. At around 65 hours, some of the smaller lymphocytes change into spindle-shaped cells and often aggregate into clumps, showing extra- and intracellular deposits of melanin. The pericardial blood-forming organ is always affected. Melanization progresses at least until 96 hours of age. Black aggregations in adults consist of cellular detritus and black masses, presumably melanin. The rectal epithelium of old tumor larvae sometimes shows nuclei reminiscent of early- and late-prophase ganglion nuclei, but of much larger size.

Ohnishi, E. Bilateral asymmetry and correlative expression of wing and hind leg in $Bd^{49\underline{1}}$. The phenotype of this Bd is very variable, ranging from wild-type to vg-like appearance, and the grades of expression of the wings of one individual are sometimes independent of each other. Besides the excision of the wing, hind legs are crumpled and this occurs only associated with heavy expression of the wing excision. This is true also with respect to both sides of one asymmetrical individual. This correlation suggests that some cooperation exists between the imaginal discs of the dorsal mesothoracic and the ventral metathoracic, rather than a mosaic nature of this mutant.

Ohnishi, E. Tyrosinase activity during puparium formation in D. melanogaster. Rapid increase and decrease in tyrosinase activity during puparium formation was observed by the following techniques. Larvae or pupae were ground in a glass mortar and made into a pulp with distilled water. This was centrifuged, and the supernatant fluid (without the lipid layer) and precipitated cell debris were measured separately by Warburg's manometer, using catechol as the substrate. Special care was taken to use individuals of exactly the same stage. Values for Q_{O2}, calculated from the oxygen uptake of the first 5 minutes, are shown in the table.

	Tyrosinase activity (Q_{O2})		
Stage	Extract	Cell debris	Sum
larva moving on the wall........	trace	trace	-
larva immobile (ca. 1 hr before puparium formation).....	8.2	14.9	23.1
prepupa with white puparium.....	12.2	15.5	27.7
prepupa 1.5 hr after puparium formation.....................	3.7	10.1	13.8
prepupa 3.0 hr after p.f.	1.6	6.4	8.0
prepupa 6.0 hr after p.f.	2.2	4.4	6.6
prepupa 12 hr after p.f.	0.7	1.4	2.1

Q_{O2} = microliter O_2/mg body weight hour

In spite of the very high activity of the white prepupa preparation by the grinding method, an almost inactive preparation was obtained by the following procedure. White prepupae were immersed in 0.75% NaCl solution and dissected by needles under a binocular microscope, with special caution not to injure the tissues, and the puparium was torn into 3-4 pieces, setting free body fluid into the NaCl solution. Remaining tissues and puparium were ground and suspended in water. This diluted body fluid and suspension were both almost inactive. When both were mixed and left to stand for 10 minutes, then separated by centrifuging, some activity appeared. Considering these and other factors, it may be concluded that tyrosinase in vivo is probably in an inactive state, and is activated by an activator of unknown nature present in the tissues. Details will be shown elsewhere.

Oster, Irwin I. Accentuation of distinctions in larval Malpighian coloration by the feeding of riboflavin.

Kikkawa (DIS-22) reported that riboflavin is the substance which gives the Malpighian tubes their coloration. Following this, a statement, of undetermined origin, has received circulation, that the feeding of riboflavin to larvae results in a deepening of the color of the Malpighian tubes. In order to study this phenomenon, I tried feeding larva with a concentration of 0.3 g riboflavin per liter of food. It was found that the larvae of flies of genotypes giving adults with phenotypically normal eye color were thereby caused to acquire a deep yellow-orange color in their Malpighian tubes. Eight wild-type stocks derived originally from very diverse sources (including Florida, Japan, and Argentina) were all found to give this deep color, as also were stocks showing the phenotypes of 21 different mutant genes not affecting eye color. Stocks with mutant eye colors gave the following results for the color of the Malpighian tubes (second column) as compared with the colors of larvae not fed extra riboflavin (first column), as previously reported by Brehme and others (see Bridges and Brehme, 1941) or as determined by us.

Eye-Color Phenotypes of Adults	Color of Malpighian Tubes of Larvae	
	Without extra riboflavin feeding	With extra riboflavin feeding
p^p, st ca, w^a, w^e	colorless	colorless
cm	very pale yellow	very pale yellow
bw, cn, cn bw, st, v	pale yellow	pale yellow
bur cn, car	pale yellow	deep orange
glass-like (DIS-25), pr, pu, ras^2, se	bright yellow	deep orange

From the above it is evident that the feeding of riboflavin accentuates the differences between the colorless and the bright yellow (= normal) tubes, and also between the colorless and certain of the pale yellows, as well as between the other pale yellows and those ordinarily bright yellow. Hence this method should be useful for the better discrimination of larvae of different genotypes, whose classification would otherwise be more difficult and uncertain. At the same time, interesting biochemical problems are raised concerning the basis of these differences in the treatment of riboflavin by Malpighian cells of different genotypes, and the relation of this to the processes occurring in the development of eye pigments.

Oster, Irwin I. An analysis of ultraviolet-induced lethals due to gene mutation.

Medvedev (1938), in a developmental analysis of 30 cases of sex-linked lethals that had arisen spontaneously and that showed no apparent changes in their salivary-gland chromosomes, found that 15 exerted their lethal effect during the pupal stage. (It is not stated to what extent the possibility of allelism among these lethals had been ruled out.) In 1939 he reported that of 12 of these same spontaneous sex-linked lethals, which killed in some prepupal stage, 2 caused death during the embryonic period, 9 during the 1st larval instar, and 1 during the 2nd larval instar. Five others among the total of 30 were observed to die at about the time of pupation. He interpreted this apparent grouping as showing three "sensitive periods"--in the embryo, the first instar, and the time of pupation. Poulson (1937 et seq.) found that deficiencies usually cause embryonic death, as might be expected in view of their involving multi-locus losses, since the earliest-acting loss would be the one whose effects were seen. Hadorn (1948) tabulated the stages of death for 38 lethals reported on in previous work of various authors.

Death was found to occur at various stages, and Hadorn interpreted the results as showing a clustering of deaths at four periods: the early embryo, the period just before or after hatching from the egg case, just before or after pupa formation, and just before or after eclosion from the pupa case. He regards "*phases of high developmental activity*" as especially subject to the action of lethals. However, since many of the lethals were known to be allelic, and many were deficiencies, the collected data which he presented are such as to provide little evidence of what the relative frequency of death at different stages would be for gene mutations.

We had at our disposal a group of sex-linked lethals which had been obtained by ultraviolet irradiation of *D. melanogaster* spermatazoa in experiments by J. T. McQuate. These had been localized by McQuate in linkage tests and had then been determined by Dr. J. I. Valencia by cytological examination of the appropriate portions of the salivary-gland chromosomes to entail no visible structural changes of the X chromosome (see Valencia and McQuate, Rec. Gen. Soc. Amer., 1951). Thus these cases represent point mutations and are suited for a study of the action of individual genes on the physiological processes involved in development. The lethals were contained in balanced stocks, with the lethal X chromosome distinguished from its homologue by the markers yellow body (y) and white eye (w). These allow the male larvae with the lethal chromosome to be recognized by their light mouth parts (due to y) and their colorless Malpighian tubes (due to w). Death in embryonic stages was shown by the presence of brownish eggs and the absence of y w larvae. When death occurred in larval stages, the instar to which viability extended could be determined by the morphology of the spiracles. The moribund larvae were observed and dissected in insect Ringer's solution.

Forty-three of the ultraviolet-induced gene-mutational lethals were subjected to the above type of study. Of these only 4 were found to cause the death of the embryo (in all cases this occurred during the *late* embryonic stage), 21 caused death during the first larval instar, none during the second larval instar (or at least this was not the limit beyond which they could not pass), 7 during the third larval instar, 8 during the early pupal period, none during the middle pupal period (with the same qualification as above), and 3 during the late pupal period. Of these 43 stocks, 36 showed no morphological abnormalities as determined by gross inspection and dissection of the dead or dying stage, while 7 exhibited some derangement which might be related to the cause of death. The locus in chromosome 1, and the characteristic effects, of these 7 lethals are as follows:

ljl: lethal jawless. 14. Dies during the first larval instar, mouthparts poorly formed and sometimes absent.

lml: melanoma-like. 1. Dies during the third larval instar; larvae at time of death have internal black and brown melanotic masses (usually one or two, sometimes as many as ten), may represent a malignancy.

lrr: lethal, ring gland rudimentary. 0.3. Dies during the third larval instar, reduction of the ring gland with associated failure to undergo the third molt; otherwise normal, may live for 15-30 days.

lte: lethal, tracheae enlarged. 0.3. Dies during the third larval instar; main tracheal tubes greatly enlarged, may lack functional posterior spiracles.

ltl: lethal, tracheae lacking. 59. Dies during the first larval instar; no evidence of main tracheal tubes, although small side branches are present.

ltr: lethal, tracheae ramified. 56. Dies during the first larval instar; main tracheal tubes are thick and have bery many side branches.

lts: lethal, tracheae stretched. 8. Dies during the first larval instar; very large larvae; all the tracheal tubes are very thin, suggesting that they do not grow at the same rate as the larvae and thus become stretched.

A consideration of our data indicates that there is a clustering of mortality at the end of embryonic life, at the beginning and end of larval life, and at the beginning and end of pupal life. These are all stages where many newly developed parts and processes become functionally active for the first time. The lethal gene may have influenced development earlier, but the effect of this did not become exerted on survival until the given part or process was called on to participate in some vital physiological function. This is well illustrated in stock *lrr*. These larvae remain small, and live about 15 days as third-instar larvae but are unable to pupate because of a reduced ring gland. However, the ring-gland deficiency already was determined during the first and second larval instars by a failure of the gland's cells to enlarge. Although the greatest developmental changes of all take place in the embryo, relatively few lethals were found to act there, doubtless because functioning of the new parts is not yet active and they are not so important for life. On the other hand, a great preponderance of lethals express themselves during the first larval instar, when the manifold physiological (nervous, secretory, muscular, circulatory, etc.) processes characteristic of larval life are required. Similarly, during the end of larval and the beginning of pupal life, new and different physiological processes must take place and thus many lethals are found which express themselves then. Furthermore, the fact that no lethals were found which act mainly during the second instar was to be expected, since that period is not characterized by radically new physiological functions but only by slight morphological changes.

In addition to the above cases of gene mutation, one case of ultraviolet-induced deficiency (bands 15A2-3 to 15C4-5, according to J. I. Valencia) and one of translocation were studied similarly. The former proved to be lethal to the embryo, like most other deficiencies, although death occurred at a late embryonic stage. The latter caused death during the first larval instar.

Prevosti, A. The vti and vli characters in a wild population of *D. subobscura* Collins.

The frequency of the characters vti (venae transv. incompl.) and vli (venae long. incompl.) has been studied in a population of *subobscura* of Barcelona, using the method of inbreeding by F_1 pair matings. The experimental cultures were reared at 25° C, a temperature almost at the upper limit for the subsistence of the species, and greater than those employed by Buzzati-Traverso and Gordon, Spurway, and Street in their studies on natural populations of the same species. The results obtained by these authors, and my own results, can be summarized as follows:

	Gordon, Spurway, and Street			Buzzati-Traverso	Prevosti
	Slough	New Forest	Studland	Belluno	Barcelona
% of wild ♀♀ with *vti* in their descendants	38.3±7.1	26.2±6.8	25.5±5.7	15.5±3.9	61.5±9.5
with *vli*	23.4±6.2	11.9±5.0	20.0±5.4	8.3±3.0	34.6±9.3

The greater frequency of these characters in the population of Barcelona may be attributed, at least partially, to the higher temperature of the experiment, for other experiments show that their penetrance increases greatly with the temperature. When eggs laid by the same females were reared at 20° C and 27° C, the average increase of penetrance with the higher temperature was from 45.9±2.6 to 87.2±2.4 per cent for vti, and from 28.4±2.2 to 89.5±5.0 per cent for vli.

vti and vli are controlled by a polymeric genotype, and its relation with temperature and therefore with the rate of development suggests a possible ecological interpretation of these characters. Its presence would be related to an adaptative limitation in the variability of the polymeric genotype controlling the rate of development. Investigations are in course to test this interpretation.

Ratty, Frank J., Jr. Lethal coverage with short duplications.

A series of 5 short duplications of the w-spl area inserted into the autosomes has been tested for ability to cover lethals in the w-spl area. One of these duplications, Dp(1,3)N264-58a, was obtained from M. Demerec; the other four were produced by irradiation in this laboratory. Two are from Canton-S + irradiation and two are from w^{m4} irradiations (see New Mutants, Report of G. Lefevre). Three of the duplications show mottling for w, indicating association with heterochromatin; and two do not. All five, however, are able to cover w-lethal or Notch mutations. Six "plain" w deficiencies have been thoroughly tested against three of the duplications: N264-58 (mottled), Dp(1;3)49a7 (mottled), and Dp(1;f?)50k11 (not mottled). The latter covers all six w deficiencies, giving fertile males. The mottled duplications cover only four of them. Forty-four Notch mutants (some also deficient for w) have been tested with the above two mottled duplications. DpN264-58 covers thirty of them, twenty-two of which give fertile males. The covered fertile males are usually non-Notch in appearance, but transmit the Notch phenotype unchanged to their daughters. In a few cases, the covered males are themselves Notch, but these are invariably sterile; however, non-Notch covered males may also be sterile. The other mottled duplication, Dp49a7, seems to be shorter than N264-58, since it covers fewer mutants, but no other difference is apparent. The remaining two duplications have not yet been tested thoroughly, but can cover some w lethals and Notch mutants, giving fertile males. Thus, there is little doubt that short duplications inserted into heterochromatin so that the visible loci, such as w, rst, and spl, all show mottling are fully capable of covering the lethal effects of deficiencies for these same loci. However, the percentage of covered males in many cases is very low. Still, no correlation between coverage and secondary nondisjunction has been noted.

Sattel, Walter Running and hopping abilities of *D. melanogaster*.

Statistical analysis of data from several authors and from my own experiments, as to the hopping and running abilities of some *D. melanogaster* wild and mutant stocks, yielded the following results. (1) There are significant differences with relation to the "age" of the stocks: "old" stocks, like Oregon (cultured since 1909), show least hopping-distance; "young" stocks (inbred since 1938) show greatest distances. Significant differences as to running velocity have not yet been established. (2) Running and hopping abilities are strongly related to the constitution of the thoracic muscles. Stocks tested were: (1) Oregon, Pavia, Coimbra, Reinsdorf, Nordhausen, Berlin, "Lamarck," Corteolana, and Florida; (2) wild stocks same as (1), and al dp b, dp, Sp J/C_2, V-ple, and vg.

Scossiroli, R. Relation between successful interspecific crossings and gene arrangements.

In crosses between *D. ambigua* females and *D. pseudoobscura* or *persimilis* males having different gene arrangements, different numbers of pupae can be recovered from the same number of parental flies per culture bottle. The Chiricahua gene arrangement seems to be the most successful in such crossings.

Semenza, L., and Barigozzi, C. Chromosomes of *Aphiochaeta xantina*.

The ganglial cells, as well as the spermatogonia, show 10 chromosomes, all telomitic with strong somatic pairing. The spermatogenesis shows a very clear pachytene, with 5 pairs. Diplotene with chiasmata. No chromocenters in the resting nuclei. Salivary chromosomes generally do not show bands.

Singleton, J. R., and Zimmering, S. Interchromosomal interference in *D. melanogaster*.

An experiment was designed to determine the extent of interchromosomal interference in the absence of inversions. Females of the constitution b cn c bw were crossed by ru h th ss males, and the F_1 heterozygous females backcrossed by Cy/b cn c bw; ru h th ss/ru h th ss males (since the homozygous b cn c bw; ru h th ss males were found to be sterile). The non-Cy offspring were classified according to crossover types. A simple Chi-square test based on some 14,000 flies indicated no obvious interchromosomal interference in this experiment. A more detailed analysis on about twice as many flies is under way.

Sobels, F. H., Kruijt, J. P., and Spronk, N. Lethality due to combined action of the genes Dichaete and eyeless-dominant.

After crossing D/+ and ci^D/ey^D flies, we found that the F_1 class D; ey^D showed an almost complete lethality of 95%-100%, whereas the ey^D class showed only a relatively slight decrease to 25%. Reciprocal crosses gave slightly different results, as the offspring of D mothers rendered some break-throughs, which have only rarely been observed with ey^D mothers; These facts might point in the direction of a maternal influence. The lethality obviously only occurs in the pupal stages, as by means of marking with ebony, egg and pupal counts, no specific mortality of the D; ey^D or ey^D classes could be observed during the larval period. About 75% of the D; ey^D animals die in an early pupal stage. The late lethal pupae are characterized by highly abnormal heads, with more reduplications of the antennae and more extreme reduction of the eyes as compared to the ey^D class. The bristle pattern is always disturbed. Extreme reduction of eye size is often correlated with reduplication of the antennae. Reduplication of the antennae causes a decreased possibility of emergence, probably by affecting the ptilinum mechanism. Sometimes cause of lethality was most evident by complete reduction of the head, the labium excepted. The same abnormalities, however, to a less extreme degree, were also observed in the few emerging D; ey^D flies.

At 16° eye size is less reduced, antennal reduplications occur less often, and in consequence the amount of lethality is lowered. Temperature treatment during 60 hours at 16°, and beginning at different periods after hatching, influences eye size and antennal reduplications and gives more or less comparable results to those found by M. Vogt (1947) for Def^{r-L} and ant.

Hence we may conclude that expressivity of eyeless-dominant may be strongly influenced by the dominant gene Dichaete; a similar but weaker effect was obtained by combining ey^D with Moire. Secondly, the developmental processes causing this lethality are dependent on temperature. In addition it may be mentioned that this lethality is much higher in males than in females and that the rest of the genotype also influences the phenomena described here.

Spiess, E. B. Recent collections in New England.

The northeastern limits of distribution for Drosophila species in North America have been rather sparsely investigated;

in fact, no large-scale collections have been made farther north than southern Vermont and New Hampshire (Spiess, 1949, Jour. N. Y. Ent. Soc.). A recent collection sent from the Mt. Desert Biological Survey, Bar Harbor, Maine, during the summer of 1950 included the following species: *affinis* (1), *algonquin* (4), *athabasca* (5), *melanogaster-simulans* (173), *melanica paramelanica* (5), *putrida* (3), *quinaria* (2), *quinaria* group (?)(1), *robusta* (2), and *busckii* (4). Except for *athabasca* and *algonquin*, all other species here are first evidence of their northern distribution. Bar Harbor is in the Transition Life Zone, but since this collection was made in mid-summer it is not too surprising to find forms like *paramelanica* and *busckii* in the population.

In October of the same year we made a collection in Lexington, Massachusetts, with the following results: *algonquin* (8), *athabasca* (25), *melanogaster-simulans* (1 female), *immigrans* (5), *busckii* (3), *putrida* (1), *sigmoides* (1), *transversa* (1), *Chymomyza amoena* (1). The presence of *sigmoides* in this collection is unusual, since its center of distribution is approximately Tennessee-Carolina and it has never been taken north of New York City. The collecting site was in birch and oak woods about a quarter of a mile from a settled area. All the other species have been taken at that time of year in the Boston area.

Spiess, E. B., Ketchel, M., and Terrile, B. A. Physiological properties of gene-arrangement carriers in *D. persimilis.* Flies from Jacksonville, California, (elev. 800') containing the Whitney and Klamath arrangements of the third chromosome have been tested for egg-laying capacity, longevity, wing-beat frequency, and wing-area dimensions. Various culture conditions and mating procedures were utilized in the latter two cases; but the basic experimental procedure throughout was of mixing strains at random in a population cage in order to get genetic heterozygosity with gene-arrangement homozygosity. All cultures were kept at 15° C. Heterozygotes for gene arrangements were F_1's of population-cage progeny. Results were as follows: (1) Egg-laying capacity: WT/WT maintains about 13-15 eggs per day for about 100 days; KL/KL starts at 13.5 for ten days, but falls off to 8-9 eggs per day for the next 100 days; WT/KL maintains a high rate of production (17-20) for the first sixty days, thereafter falling off faster than homozygotes. In senescence (100-170 days from pupa) all three types give equal rates, a fact which might be expected since natural selection would have built up genetic combinations for high production in early life. (2) Both homozygotes survive equally well under these experimental conditions, but heterozygotes have a significantly lower mortality rate. All three have long-lived individuals which survive until about 170 days. (3) Wing-beat frequency (using a stroboscopic method perfected by Williams and Chadwick): WT/WT, 11,080 beats per minute; KL/KL, 9170 beats per minute, WT/KL (from WT mothers), 11,410; WT/KL (from KL mothers), 11,090. All standard errors are less than 100 beats per minute. (4) Wing-area dimensions for these types are WT/WT, 2.60 mm^2; KL/KL, 3.16 mm^2; WT/KL (WT mother), 3.07 mm^2; and WT/KL (KL mother), 2.76 mm^2. Standard errors are less than 0.04 mm^2. A "stroke-energy" index proportional to the kinetic energy given to the air by the wing beat (Reed, Williams, and Chadwick, Genetics, 1942) can be applied to determine which type is expending the greatest amount of energy. This index is as follows for these zygotic types: WT/WT, 762; KL/KL, 830; WT/KL (WT mother), 1336; WT/KL (KL mother), 907. By varying the culture conditions and mating procedures in different ways, wing dimensions and wing-beat frequencies may be caused to vary rather considerably; but in all cases so far heterozygotes have had a higher "stroke-energy" than either homozygous type, although in some cases not significantly higher. These are being retested at present.

The heterosis exhibited here is not the result of crossing after inbreeding. Strains of flies are maintained by mass matings, so that genetic heterogeneity is encouraged. The limits of heterosis due to crossing strains should already be accounted for by using the population cage, so that heterosis exhibited is clearly due to heterozygosity of the third chromosome. These tests can be used to analyze the variation involved in a gradient in relative frequencies of gene arrangements up an altitudinal transect in the Sierra Nevada.

Spurway, H. D. subobscura is very well adapted for prolonged experiments. A single pair transferred to a fresh bottle daily will often give 600 to 800 imagines, and will continue breeding for over a month. Research on the possible effect of parental age is under way, and it is hoped to construct a fertility table.

Tattersfield, F., Kerr, R. W., Taylor, J., and Kerridge, J. R. Resistance to the toxic effects of DDT. Work has been actively prosecuted to ascertain whether resistant strains of D. melanogaster (Rothamsted var.) could be obtained by repeated sprayings with DDT suspensions. A resistance of 1.5 to 3 times that of the original parent strain has emerged. The physiological and morphological characters are being studied. The effects, on resistance to DDT and CO_2, of age of individual and of parents, and of certain environmental factors, are being examined. The maximum resistance to DDT has been found, by R. W. Kerr, to occur at an age of the adult fly of about five days, and this is correlated with a higher respiration rate (Rothamsted wild-type). A "hybrid" stock supplied by B. J. Harrison, Bayfordbury, Hertford, England, is now under experiment.

Ulrich, Hans. Killing of Drosophila eggs by partial X-raying. Single sectors 0.1 mm in length, representing one-fifth of the Drosophila egg (average total length, 0.52 mm), were separately X-rayed at different ages of the eggs with a dose of 1000 r (217 r/min, 50 kv, 2 ma). The resulting percentages of non-hatching (i.e., killed) eggs were used as index of radiosensitivity of the several sectors. Eggs irradiated when 15 to 30 minutes old show a definite, high maximum (66.5% corrected) in the second fifth counting from the anterior pole, the middle fifth giving lower (24.2%) and the three other fifths very low percentages (3.5% to 5.4%). This characteristic distribution of radiosensitivity agrees well with the developmental state (according to Rabinowitz et al.). The egg when 15-30 minutes old contains either the two pronuclei or 2 to 4 cleavage nuclei, lying mainly in the second fifth. The high susceptibility of this sector may be due to the presence of these nuclei; the mean susceptibility of the middle fifth may be due to extending of nuclei to this section, but also to the method used in localizing the irradiation in this fifth. The fifths 1, 4, and 5, containing no nuclei, are not sensitive, This result contributes to the problem of whether nucleus or cytoplasm is responsible for the death of cells after irradiation. In older eggs (1-2, 2-3, up to 5-6 hours) radiosensitivity is distributed throughout the egg, but not equally, the maximum remaining in the second fifth. But in general the maximum is not so pronounced as in youngest eggs. The middle fifth gives only a little lower, the other fifths definitely lower percentages than the second fifth. The killing rates after partial X-raying of eggs older than 2-3 hours are low throughout. This agrees with the result of total X-raying, demonstrating a rapid decrease of radiosensitivity in older stages of embryological development.

Ulrich, Hans. Sensitive periods and egg-regions in production of the modification "abnormal abdomen" by X-raying eggs of D. melanogaster.

When X-raying Drosophila eggs at different stages with different doses, the author did not find the sensitive periods of the modification "abnormal abdomen" (aa) found by Henke and Maas when treating eggs with high temperature. In special experimentw with 4- to 5-hour eggs, percentage of killed individuals (eggs, larvae, and pupae) increased with dose more rapidly than did percentage of flies showing aa. With a dose of 800 r, the rate of aa reached its maximum of about 20% (few animals), while 93.5% of individuals died. This suggested that higher rates of aa cannot be obtained when eggs are X-rayed totally, because the doses required to produce them are lethal.

If sensitivity to the effect of X-rays in producing aa is distributed in another manner in the egg than sensitivity to lethal effect (see preceding note), it should be possible by means of partial X-raying to apply higher doses (while shielding the region highly lethal-sensitive and exposing the highly aa-sensitive one) with the result of obtaining a lethal rate much lower than 100% and a percentage of aa higher than reached before by total X-raying. Eggs at the ages of 15-30 minutes, 1-2 hours, 2-3 hours, etc., up to 7-8 hours were partially treated with a dose of 1000 r. When applied to total eggs of, say, 4-5 hours, this dose kills 99.9% of individuals; and when applied to single fifths of 4-5-hour eggs, it kills 38% to 68%, the percentage depending on the position of the treated fifth. The resulting percentages of aa after partial X-raying (see table) demonstrate a first low maximum at 1-2 hours in the middle fifth of the egg. With increasing age the maximum decreases and migrates towards the posterior pole of the egg, coming up to a new high peak at an age of 4-5 hours in the last two fifths of the egg. With further increasing age the maximum decreases and finally disappears. The method of partial X-raying makes it possible to detect sensitive periods not to be found when eggs are totally irradiated, and, moreover, to find sensitive regions of the modification "abnormal abdomen".

Percentages of Flies with Abnormal Abdomen

Age of eggs when X-rayed	No. of the treated fifth 1	2	3	4	5
15-30 min.	0.6	4.2	1.7	1.5	0.8
1-2 hours	2.8	10.8	27.6	14.6	1.3
2-3 "	0.6	0.0	7.1	7.8	2.8
3-4 "	1.0	1.3	3.1	11.3	13.5
4-5 "	0.6	0.8	0.7	38.0	46.5
5-6 "	1.7	0.4	0.0	7.9	7.0
6-7 "	3.5	1.2	0.0	9.6	11.8
7-8 "	0.7	0.7	0.0	2.8	0.7

Valencia, J. I., and Valencia, R. M. The ineffectiveness of extra heterochromatin in influencing mutation rate in the female.

In the course of a recent experiment on the X-ray induction of mutations at specific loci in the female (Rec. Gen. Soc. Amer. 18: 105, 1949), it was observed that the spontaneous mutation rate (for both visibles and lethals) was unusually high in the stock used. Since this stock (b123, "plond," DIS-22) contained an extra Y chromosome (of sc.Y^1 type), the question arose whether extra heterochromatin might be influencing mutation rate. The following experiment was carried out to check this hypothesis, following a genetic scheme devised by H. J. Muller.

Spontaneous recessive lethals were looked for in a particular X chromosome (y sc^8 B f In49 v) among the progeny of females having this X and either having (experimental series) or not having (control series) an extra Y chromosome. The control and experimental females of the generation (called P1) which was tested for its spontaneous mutation frequency were sibs of each other and did not differ genetically from each other in any systematic way except by the presence or absence of a sc.Y$^{\underline{1}}$ chromosome (recognized by their being non-yellow or yellow). The crosses of flies, called P$_o$ (see the figure), that produced these P$_1$ females were designed in such a way as to make possible the elimination of all pre-existing lethals, to equalize the experimentals and controls by randomization of the autosomes, to make each of the many experimental and control lines isogenic with one another for the two X chromosomes (in the P$_1$ generation, in which lethals occurred), and to allow identification, in each generation, of all female combinations of X's and Y's, whether formed by disjunction or by nondisjunction. Since disjunctionally produced males from the crosses could have either one or two Y chromosomes, the male parents in each generation (except the last, where this did not matter) were taken from attached-X stocks.

We tested 3864 experimental and 3864 control chromosomes. In the former, 12 lethals (.31%), and in the latter 13 lethals (.33%) were observed. Thus we conclude that, in females at least, extra heterochromatin does not influence the spontaneous lethal mutation rate.

Crossing scheme used in lethal experiment. Phenotypes in parentheses.

P$_o$
y sc^8 B f In 49 v
y w ct . Ys
sc . Y$^{\underline{1}}$
(B ♀)
x $\underline{1}$ (about 50 individual matings)
y ct f . Ys
sc . Y$^{\underline{1}}$
(ct f ♂)

Experimental — sisters — Control

P$_1$
y sc^8 B f In 49 v
y ct f . Ys
sc . Y$^{\underline{1}}$
(f B ♀)
(several from each P$_o$ vial which was nonlethal)
x
y w ct . Ys
sc . Y$^{\underline{1}}$
(w ct ♂)
(from stock)
x
y sc^8 B f In 49 v
y ct f . Ys
(yf B ♀)
(several from each P$_o$ vial which was nonlethal)

F$_1$
y sc^8 B f In 49 v
y w. ct . Ys
(y B ♀)
(3 from each P$_1$ vial which was nonlethal)
x
y ct f . Ys
sc . Y$^{\underline{1}}$
(ct f ♂)
(brothers)
x
y sc^8 B f In 49 v
y w ct . Ys
(y B ♀)
(3 from each P$_1$ vial which was nonlethal)

F$_2$ Look for lethals — Look for lethals

Confirm any found by getting F$_3$

von Brandt, H., and Höhne, G.
Mutagenic action of some chemicals.

The following compounds have been tested for mutagenic action in *D. melanogaster*: (a) ethylurethane, dissolved in 0.96 KCl, (b) tri-(2-chlorethyl)amin, dissolved in citric acid and 0.96% KCl, (c) p-dimethylaminoazobenzene, dissolved in sesame oil, (d) 2-methyl-1,4-naphthohydroquinone, dissolved in 0.96% KCl, and (e) 4,4'-stilbendicarboxamidine, dissolved in 0.96% KCl. In all experiments sublethal doses of the chemicals were administered by means of injection of imagines, except that in (c) we also exposed imagines to a sesame oil aerosol. The wild-type males treated (Berlin wild) were tested for sex-linked recessive lethals by the ClB or Muller-5 method. The mutation rates of the control groups (treated only with KCl, NaCl, citric acid, or sesame oil) varied from 0.2 to 0.5%. The following table shows the results. Apart from the well known mutagenic agents ethylurethane and tri-(2-chlorethyl)amin, there were no significant increases in frequency of lethals after treatment with (c), (d), and (e).

Compound tested	No. chromosomes tested	No. lethals	% lethals
(a) Injection - 0.3%	1394	22	1.58
(b) Injection - 0.10	172	3	1.74
- 0.03	353	19	5.38
- 0.01	1189	38	3.20
(c) Injection - 2.3%	3220	22	0.68
Aerosol - 2.3%- 48 hours	2298	4	0.13
(d) Injection - 1.0%	1496	4	0.27
-0.01%	1245	10	0.80
(e) Injection - 1.0%	662	4	0.60
Control group (Injection of NaCl, KCl, or citric acid)	3741	14	0.37

Waddington, C. H. Selection of the genetic basis of an acquired character.

Developing flies of a wild-type strain originally collected in Edinburgh were given a temperature shock by being placed at 40° C for four hours at about 21-23 hours after pupation. A crossveinless phenocopy was produced with a frequency of about 40%. One selected line was started from the crossveinless flies, the phenocopies being bred from in each generation; in a second selected stock, breeding was from those which did not show the phenocopy. After 15 generations, the frequency of phenocopies had become over 90% and under 10%, respectively. In the twelfth generation of the upward-selected stock, crossveinless flies appeared even among individuals to which the temperature shock had not been given. When these were bred from, the condition was certainly inherited, probably by a gene of incomplete penetrance whose behavior has not yet been fully worked out. Thus the crossveinless condition, initially produced as a response to an environmental stimulus, has during the course of selection picked up a genetic basis which enables it to appear in the absence of the stimulus.

Wallace, Bruce, and Demerec, Rada A test for translocation mosaics in Drosophila sperm exposed to nitrogen mustard aerosol.

In a recent article (B. Wallace, Dominant lethals and sex-linked lethals induced by nitrogen mustard. Genetics 36: 364-373, 1951) it was suggested that the genetic test for translocations (y; bw; e) may fail to reveal mosaic males carrying translocations in one portion of their sperm and normal chromosome complements in the other. Such males would remain undetected because all expected classes of individuals would be present in the final (F_2) cultures although not in equal proportions. Since tests for translocations are generally made in vials, the distortions in the relative proportions of the various classes would be ascribed to chance. It was conceivable that the low frequency of recoverable translocations after nitrogen mustard treatment could be partially explained on this basis. The hypothesis has been subjected to experimental test, but the results are inconclusive. They may be interesting, however, in other connections.

In two experiments, 465 sperm from 57 treated males (2% aqueous solution of nitrogen mustard; 4-6 hours' exposure) were tested for translocations. Three were found (0.6%; limits of the 95% confidence interval, 0.2%-1.9%) F_2 males were retested for translocations. If any of the original 465 sperm carried an unstable condition that resulted in mosaicism for a translocation, some of the F_2 males descended from that sperm should carry a translocation undetected in the F_1, while the others should be normal. 4342 F_2 males were tested--an average of 9.3 males for each of the original 462 sperm that gave negative F_1's. No translocations were detected in this generation. The highest frequency of mosaics compatible with these observations is 0.6% (95% confidence).

To explain by mosaicism the low frequency of translocations (relative to X-ray treatments giving similar sex-linked-lethal frequencies) after exposure to nitrogen mustard, one must assume that mosaics are induced with a higher frequency than nonmosaic translocations. Since the 95% confidence interval of the frequency of mosaics detected in the F_1 spans the upper limit of the 95% confidence interval of the frequency of translocations in the F_2, we must conclude that the test has failed and no conclusion can be reached concerning the hypothesis to be tested. The available data, however, may be sufficient to subject other hypotheses to test and for such hypotheses they are perfectly valid.

Wette, Reimut Production of phenocopies by chemical substances.

Rapoport reports induction of phenocopies by 1,4-derivatives of benzene in *D. melanogaster*. Of several substances being tested in our experiments with dead-yeast cultures, hydrochinone has so far yielded a clear effect: 90% of the F_1 flies showed monostrophic asymmetries of the twisted type in the abdomen.

Yoshida, Y. "Conditioned lethal."

In *D. melanogaster*, a certain mutation provisionally named "conditioned lethal" was found in the second chromosome of a stock of bw dp, during an experiment using the stocks bw/Cy and bw dp. But it seems that "conditioned lethal" is independent of bw and dp. The homozygous stock of "conditioned lethal" is viable and fertile, but "conditioned lethal/Cy" always gives only "conditioned lethal/Cy," and never the homozygote of "conditioned lethal."

In the offspring of "conditioned lethal/Cy" females x "conditioned lethal homozygous" males, the homozygote is lethal. In the reciprocal cross, however, the homozygote is semilethal. In the offspring of "conditioned lethal/Cy" females x wild-type males, the heterozygote of "conditioned lethal" is semilethal, but viable in the reciprocal cross. It seems that the homozygote of "conditioned lethal" is generally viable and fertile, but that it is lethal or semilethal with certain genetic factors. The heterozygote of "conditioned lethal" is semilethal with these factors, which are being analyzed as to whether they are simple or complex, and whether they are chromosomal or cytoplasmic.

TECHNICAL NOTES

Anders, Georges, and Schnitter, Marco A method of mass investigations in Drosophila eggs. Eggs are collected, dechorionated with sodium hypochlorite, and washed in distilled water, after which they are placed in rows on a glass slide and allowed to adhere by drying. Then they are treated in the following way:

1. Prick each egg gently with a steel needle and let the exuding content dry.
2. Submerge the slide for several minutes in a dish containing ca. 0.5% collodion in a mixture of equal parts of ether and absolute alcohol.
3. Allow to dry for several seconds and put in 70% alcohol for 5 minutes.
4. Let remain for 10 minutes in distilled water.
5. Transfer the slide to 1 N HCl at 60° for 6 minutes.
6. Wash for several minutes in distilled water.
7. Stain in Feulgen dye for 2 to 3 hours.
8. Wash for 1 hour in running tap water.
9. Run up through alcohols to Euparal.

In order to avoid plasmal reaction the slide may be treated before hydrolysis with 96% alcohol in the usual way. Moreover, after dying, the eggs may be washed with SO_2-water to prevent staining of cytoplasm. Both treatments we found to be unnecessary for current work. The method is useful for testing fertilization in young eggs and for determination of the stage at which embryos belonging to a lethal genotype die.

Clancy, C. W. "Seeding" cultures with Fleischmann's New Dry Yeast. I find a saltcellar (shaker) very convenient for distributing the few granules of this material required to properly inoculate a vial or bottle with live yeast.

Green, M. M. Rapid preparation of cornmeal-agar medium. We have found that the use of a pressure cooker of the type commonly used in home canning facilitates the preparation of the usual cornmeal-agar medium. A measured amount of water (according to the volume of medium to be prepared) is brought to a boil in the uncovered cooker. The other ingredients--agar, soaked cornmeal, molasses, brewers' yeast, etc.--are added, the cooker is covered, and the material autoclaved for 10 minutes. The resultant medium is ready for pouring. Since the water loss by this procedure is small as compared to the loss during the usual methods of preparing media, the amount of water used is decreased by 10%.

Herskowitz, Irwin H. Grid-smear technique for electron microscopy of salivary-gland chromosomes. Several techniques have been described for the preparation of Drosophila salivary-gland chromosomes for electron microscopy, employing replicas (casts), stained sections, and stained smears. This note describes a new and very simple technique for such preparations. Full-grown larvae of D. melanogaster are placed in 60 per cent acetic acid, and the salivary glands removed. After 10 minutes in this solution the glands are transferred to a 200-mesh nickel grid previously coated with a water-floated film of parlodion. Another coated grid is put on top of this, the grids placed between two slides, and the glands crushed by thumb pressure. After the squashing, the two grids are separated with a needle and permitted to dry in air for a few minutes. The tissue is then ready for microscopy and remains in excellent condition for months. The grid-smear method is time-saving, permits microscopy of unstained tissues, and eliminates the necessity of subjecting the material to various chemical and physical agents, which might cause distortion in addition to the original fixation.

Herskowitz, Irwin H., and Burdette, Walter J. Preparation of permanent aceto-orcein smears.

Permanent aceto-orcein smears of Drosophila salivary-gland chromosomes may be prepared routinely by means of the following technique. Salivary glands are removed from larvae during the third instar after they have been placed in 60% acetic acid. After 10 minutes in this solution the glands are crushed in the usual manner between a slide and a coverslip previously covered lightly with albumen. The coverslip is floated off with the stain, consisting of 2% orcein in 60% acetic acid. Most of the tissue adheres to the coverslip, and contact with the stain is necessary for only a few seconds. The coverslip is then mounted on a clean slide bearing a small drop of light Karo corn syrup. Excess Karo is removed by pressure and the preparation permitted to harden. By covering the margins of the coverslip with Clarite or a similar mounting medium, such preparations are made waterproof.

There are several advantages of this method besides simplicity. The acetic acid induces sharp definition of the bands and, since it is used alone, permits excellent chromosome spreads. The Karo washes away excess stain and leaves the background of the chromosomes clean. Moreover, with simple modifications, this method permits one to retain ordinary preparations for an extended period. Salivary chromosomes are particularly well seen using a 1.25-mm dark M phase objective.

Mittler, Sidney Medium for rearing yeasts that do not require amino acids or vitamins.

In attempt to control the nutrition of *D. melanogaster*, yeasts were selected that could grow on a vitamin-amino acid-free medium. The following medium was employed:

Agar	15 gm	Nacl	0.5 gm
$C_6H_{12}O_6$	30 gm	$MnSO_4$	0.5 gm
KH_2PO_4	1 gm	$MgSO_4$	0.5 gm
$NaKC_4H_4O_6$	8 gm	$FeSO_4$	0.5 gm
$(NH_4)_2SO_4$	2 gm	H_2O	1000 cc
$CaCl_2$	0.5 gm		

When this medium is inoculated with a yeast that can live in the absence of vitamins or amino acid, practically all the nutrition obtained by the flies is from the yeast. If one uses a yeast like *Hansenula anomala* NRRL^{365} with the above minimal medium at a temperature of 24° C, one has a set of conditions that can be reproduced. With the cornmeal-molasses medium there are probably as many variations possible as there are research workers. In the study of penetrance and expressivity it is of utmost importance to have nutrition as well as temperature under control.

Rosin, S. The position of the wings of killed drosophilae.

In flies that have been killed by overetherizing the wings are maintained in a vertical position, so that some bristles cannot be easily seen. In order to study bristle pattern in fixed material, 70%-80% alcohol heated to 70°-80° C is poured over the well-etherized (but not overetherized) flies. By this fixing method we get a more or less normal position of wings.

Stone, P. C., and Zimmering, S. An effective mite control. The Drosophila lab at Missouri had for many years been heavily infested with mites. Complete control has been achieved by use of a new organic mitecide, Aramite-15-W, so that for the past three months no mites whatever can be found. Adult flies were transferred to a fresh food vial together with about 150 mg (the amount that can be held on a penny) of the full-strength Aramite (15% active ingredient), and shaken so that they were well covered with the powder. Cotton plugs were also dusted with Aramite. As a routine, this process was carried out for two generations. Used in this way, Aramite will kill the hypopus as well as other stages of mites, but does not seem to harm the adult drosophilae or affect their fertility. It is important that there be no free water on the walls of the vials, as Aramite will then make a paste with the water, which kills flies. This procedure was carried out in a separate room, the empty lab being, in the meantime, fumigated with a commercial mixture of 3 parts ethylene dichloride to 1 part carbon tetrachloride, about 15 pounds per 1000 cubic feet being used. Aramite 15-W is obtainable from the U. S. Rubber Co., Naugatuck Chemical Division, Naugatuck, Connecticut.

Wallace, Bruce Estimating the size of experimental Drosophila populations. In a study of either the ecology or the genetics of a population, one of the important factors is population size. Determining the number of adults in a Drosophila population by etherization and counting is a laborious task (the number may exceed 10,000), which disrupts, with possible selective effects, the continuity of a population. The result obtained is hardly more than an estimate, because several hundreds of the flies remain uncounted in the cage and moribund flies are included in the final figure. Any technique that gives a rapid estimate of the number of adults would be a useful one.

An attempt at estimating the number of flies by sampling "fly specks" has been made with some success. The experiment completed dealt with the relation between a known number of adult flies in a population and the number of specks obtained on a sampler exposed to the population for certain periods of time. The cage used was one of Lucite and screen, 18 inches long by 5 1/2 inches wide by 4 1/2 inches high. The sampler was a glazed porcelain cylindrical electrical insulator 3/4 inch long and 5/8 inch in diameter. It was mounted on a glass rod 7 inches long, which was inserted into a rubber stopper. Samples of specks were taken by projecting the sampler through a hole in the small end of the cage and plugging this hole with the stopper on which the sampler was mounted. The sampler, consequently, was suspended equidistant from the top, bottom, and sides of the cage, 7 inches from one end. The number of specks obtained was determined under a low-power binocular microscope merely by counting and simultaneously touching each speck with the point of a pen. Specks, even when overlapping, were easily distinguishable against the white porcelain background.

The results of the experiment can be tabulated as follows:

Day	No.new flies added*	Total flies in cage#	No.specks (4-hour exposure)**	No.specks (17-hour exposure)##
1	674	674	21	32
2	688	1362	24	53
3	1140	2502	48	185
4	1496	3998	37	142
5	2213	6211	48	270
6	1858	8069	102	407
7	1526	9595	106	386

* Obtained each day from a series of culture bottles.
# Assuming, erroneously, that no deaths occurred.
** Sampler exposed from 12:30 p.m. to 4:30 p.m.
## Sampler exposed from 4:40 p.m. to 9:40 a.m.

The slope and error of the slope of the regression of specks on flies during the four-hour interval was .0095 ± .0005 (error = 5.3% of the slope). For the 17-hour interval the corresponding figures were .0421 ± .0054 (error = 12.8% of the slope). The ratio of the slopes, .0421/.0095, was 4.43; and the ratio of the lengths of exposure, 17/4, was 4.25.

These data indicate that this is a technique that can be used for determining the relative number of flies in a population at frequent intervals without disturbing the population unduly. Whether it can be used to compare one population with another or to estimate actual numbers of flies under various conditions is not known.

Wilson, L. Deterioration of brewers' yeast as a factor in comparative growth studies on Drosophila.

A supply of dry brewers' yeast kept in a dark bottle under ordinary laboratory conditions eventually failed to support growth in Drosophila. The yeast was purchased in October, 1949, and used for routine sterile growth studies at a concentration of 4% until August, 1950. Suddenly at that time larvae in all experiments died before the first moult. Increasing the concentration of yeast did not prevent the deaths, but a high proportion lived in 0.75% yeast. These results suggested a vitamin deficiency which was not great enough to prevent development of the slow-growing, inadequately fed flies. To test this hypothesis the yeast was supplemented with different brands of B-complex vitamins in a concentration sufficient to supply 200 micrograms of thiamin per 100 milliliters of medium. Perfectly normal growth resulted. No growth occurred if the vitamins were autoclaved in distilled water before being added to the culture medium. It has been possible to test the rate of deterioration of strains of yeast by periodically checking the rate of growth of the larvae and the time of occlusion of the adults. A strain of yeast purchased in February, 1951, and kept continuously in the refrigerator began to show deterioration in September, 1951. It is obvious that in comparative growth studies where yeast is used in the media, great care must be exercised to insure a yeast of constant nutritional value.

D. G. Catcheside is leaving Cambridge to take up a post at the University of Adelaide, Australia (see Directory), where he expects to arrive about the middle of January. He is taking his collection of Drosophila stocks with him.

Robert C. King has received an appointment at Brookhaven National Laboratory, where he will continue his work on induction of mutations in Drosophila with the aid of radioisotopes.

Sarah Bedichek Pipkin is at present in the Caroline Islands of the West Pacific (see Directory). Her stock list was received too late for inclusion, but she writes that she is doing some experimental work and also some collecting.

Öistein Strömnaes has moved from the University of California, Berkeley, to Arvelighetsinstituttet, Universitetet, Oslo, Norway, where he is studying the inheritance of susceptibility to X-ray-induced mutations under the sponsorship of the Norwegian Research Council.

The IXth International Congress of Genetics will take place at the end of August, 1953, in Bellagio (Como), Italy. The Secretary is Professor C. Barigozzi, ISTITUTO DI GENETICA, UNIVERSITÀ DI MILANO, VIA CELORIA 10, MILAN, Italy.

University of Melbourne, Australia. A genetics laboratory has recently been established, and is engaged in a research program on the population genetics of Australian species of Drosophila.

University of Queensland, Australia. The teaching of genetics is being expanded and a research project on the systematics of Queensland species of Drosophila has been entered upon. The Department would appreciate any back numbers of DIS that may be available, and reprints in all fields of genetics. Please address care of W. B. Mather.

Institute of Animal Genetics, Edinburgh. A recent administrative reorganization will do away with the distinction between the University Department of Animal Genetics and the Genetics Laboratory of the Animal Breeding & Genetics Research Organisation of the Agricultural Research Council. In future the finances of the second body will be passed over to the University, and all workers in the Institute will be considered as belonging to the Department of Animal Genetics, Edinburgh University.

The addresses of workers mentioned in DIS-23 and DIS-24 who have since left the Institute are as follows:

Callan, H. G., now Professor of Zoology, University of St. Andrews, Scotland.
Lamy, R., Department of Genetics, University College, London.
Mehtab, M., Government College for Women, Lyallpur, West Punjab, Pakistan.
Rendel, J. M., C.S.I.R.O. Genetics Section, Department of Zoology, University of Sydney, Australia. (Seconded for 3 years).
Sang, J. H., Poultry Research Centre, West Mains Road, Edinburgh.
Tantawy, A. O., Department of Agriculture, Farouk Ist University, Alexandria, Egypt.

A "Type Culture Collection of Drosophila" has been established at the Institute of Genetics of the University of Pavia, under the auspices of the International Union of Biological Sciences and with partial financial support by UNESCO. It is intended to keep a large collection of wild type and mutant strains of species other than D. melanogaster. The lists of available material will appear in DIS. More detailed information on the available strains can be obtained directly from the Pavia Institute. Research workers in the field are kindly requested to send material that they wish to be preserved.

A new Center of Genetics at the University of Barcelona, Spain. A Center of animal and human Genetics, supported by the Consejo Superior de Investigaciones Científicas has been organized at Barcelona. This Center includes a Department of Drosophila Genetics, headed by Dr. Antonio Prevosti, who would appreciate any back issues of DIS that might be available, and reprints in all fields of genetics.

MATERIALS REQUESTED OR AVAILABLE

Walter J. Burdette needs a stock of D. melanogaster with a gene for high mutation rate. Information concerning such a stock will be appreciated.

A. A. Buzzati-Traverso is engaged in the study of interspecific crossings in the obscura subgroup. Please send available strains of D. ambigua as well as of recently discovered species belonging to this subgroup, provided of course that holders of such stocks do not already plan to use them for the same purpose.

M. M. Green would appreciate receiving any new occurrences of mutations, induced or spontaneous, at the v locus in D. melanogaster.

D. D. Miller of the University of Nebraska (Zoology Department) would appreciate receiving recently derived strains (or wild caught females from which strains might be started) of D. athabasca (especially from western North America), D. azteca, and D. narragansett.

Antonio Prevosti of the University of Barcelona (see Personal and Laboratory News) would appreciate receiving reprints in all fields of genetics.

The Genetics Laboratory of the University of Melbourne, Australia would like to receive reprints of work on population genetics.

The Department of Zoology of the University of Queensland (see Personal and Laboratory News) would appreciate receiving reprints in all fields of genetics. Please address care of W. B. Mather.

The "Type Culture Collection of Drosophila," Pavia, Italy (see Personal and Laboratory News) is interested in collecting European species. Please send available material, stating locality, altitude, and date of collection, as well as special breeding requirements, if any.

Reprints of the following papers by Margaret E. Hoover may be obtained by writing to M. Demerec, Carnegie Institution, Cold Spring Harbor, New York:

A case of inversions in tandem in the X chromosome of D. melanogaster. Genetics 22: 195, 1937. (Abstract)

A tandem inversion in Drosophila melanogaster. Genetics 22: 634-640, 1937.

Correlation between inversion length and synaptic attraction in salivary chromosomes of Drosophila melanogaster. Genetics 22: 195-196, 1937. (Abstract)

Cytogenetic analysis of nine inversions in Drosophila melanogaster. Zeitschrift für induktive Abstammungs-und Vererbungslehre Bd. LXXIV, Heft 3/4, pp. 420-434, 1938.

Irwin H. Herskowitz, Editor

D. = *Drosophila*; D.m. = *Drosophila melanogaster*

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(Alphabetically arranged according to country, city, laboratory. * Signifies that the entry was copied from DIS-24 because no new statement was received.)

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Anders, Georges. Graduate student. Developmental genetics, lozenge.
Bertschmann, May. Graduate student. Influence of chemicals on development.
Bucher, Nelly. Graduate student. Development of gonads.
Burla, Hans. Ph.D. Assistant. Drosophila species.

Chen, Pei-Shen. Ph.D. Assistant. Physiology and development.
Gloor, Hans. Ph.D. Dozent. Developmental genetics.
Hadorn, Ernst. Ph.D. Professor. Developmental genetics, lethals, influence of chemicals.
Oprecht, Eva. Graduate student. Parasitic wasps of Drosophila.
Perron, Rolf. Assistant. Development of Drosophila mites.
Staiger, Hans-Rudolf. Ph.D. Assistant. Cytology.
Zollinger, Elisabeth. Graduate student. Subobscura: cytogenetics.
Zwicky, Karl. Assistant. Physiology.

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Ames, Iowa
Iowa State College, Department of Genetics

Fryxell, Paul A. M.S. Linkage, heterosis.
Fung, Sui Tong. M.S. Melanogaster: physiological effects of sex genes, chromosome reorganization.
Gowen, John W. Ph.D. Melanogaster: crossing over, gene structure and physiological action, heterosis.
Grahn, Douglas. M.S. Linkage, gene action in development.
Griffing, J. Bruce. Ph.D. Linkage heterosis.
Kopf, Kenneth. Linkage, physiological effects of genes.
O'Mara, Joseph G. Ph.D. Cytology.
Poirier, Rolland P. M.S. Linkage.
Stadler, Janice. M.S. Melanogaster: agents for mutations, heterosis.
Vincent, Walter S. M.S. Cytochemistry.

Amherst, Massachusetts
Amherst College, Department of Biology

Evans, Alice T. (Mrs.) B.S. Curator of stocks and research assistant.
Ives, P. T. Ph.D. Research Associate. Melanogaster: speciation, mutation, gene action, population studies.
Levine, R. P. Ph.D. Instructor. Pseudoobscura: population genetics.
Plough, H. H. Ph.D. Professor. Mutation and gene action. (On leave until July, 1953.)
Young, William J. B.A. Graduate assistant. Position effect.

Army Chemical Center, Maryland
*Medical Division

Bodenstein, Dietrich. Virilis, melanogaster: developmental physiology, metabolism.
Chadwick, L. E. Ph.D. Melanogaster, virilis; physiology of flight.
Hassett, C. C. Ph.D. Melanogaster: physiology.
Noland, J. Ph.D. Melanogaster; nutrition.
Sacktor, B. Ph.D. Melanogaster, virilis; respiratory metabolism.

Austin 12, Texas
The University of Texas, Department of Zoology

Bruneau, L. H. Research Assistant.
Clayton, Frances E. Ph.D. Instructor. Radiation effects.
DeBusk, A. G. Dreyman Fellow. Gene action.
Dudgeon, Edna. Research Assistant. Radiation effects.
Haas, F. L. Ph.D. Research Scientist. Radiation effects.
Haddox, C. H. Ph.D. Research Scientist. Radiation effects.
Heed, William. Research Assistant. Speciation.
Moorhead, P. S. Research Assistant. Speciation.

Oliver, C. P. Professor. Gene action.
Patterson, J. T. Professor and Director. Speciation.
Stone, W. S. Professor. Speciation & gene action.
Wagner, R. P. Associate Professor. Gene action.
Wasserman, A. Research Assistant.
Wasserman, M. Research Assistant.
Weinberg, R. Research Assistant. Speciation.
Wheeler, M. R. Instructor. Taxonomy & speciation.

Baltimore, Maryland
The Johns Hopkins University, Department of Biology

Erk, Frank. A.B. Graduate student. Melanogaster: action of selection on translocations in artificial populations.
Glass, H. Bentley. Ph.D. Melanogaster: population genetics of erupt and suppressor-erupt; gene action of suppressor-erupt; effects of mutagens on females.
House, Verl L. Ph.D. Melanogaster: development of venation.
Novikoff, Jane. A.B. Graduate student and research assistant; effects of mutagens on females.
Plaine, Henry L. A.B. Graduate student. Melanogaster: gene action of suppressor-erupt.
Ritterhoff, Rebecca K. A.B. Research assistant. Effects of mutagens on females.

Baltimore, Maryland
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Baker, Percy H. Ph.D. Associate Professor. Melanogaster: mechanism of gene action.

Bar Harbor, Maine
Roscoe B. Jackson Memorial Laboratory

Griffen, A. B. Ph.D. Melanogaster: position effect, cytogenetics.
Kaliss, Nathan. Ph.D. Melanogaster: physiological genetics.

Berkeley 4, California
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Buzzati-Traverso, Adriano Sc.D. Visiting Professor, fall semester.
Carson, Gweneth L. M.A. Research assistant.
Goldschmidt, Richard B. Ph.D. Professor Emeritus.
Hannah, Aloha. Ph.D. Research associate.
Hexter, William. A.B. Graduate student.
Hildreth, Philip. M.A. Research assistant.
Hochman, Benjamin. A.B. Graduate student.
Lewis, Herman W. M.S. Graduate student.
Lin, Teh P. M.A. Research assistant.
Mohler, James D. M.A. Graduate student.
Piternick, Leonie Kellen (Mrs.) Ph.D. Independent investigator (Assistant Professor, Mills College).
Shaw, Richard. M.A. Graduate student.
Stern, Curt. Ph.D. Professor. (At Laboratoire de Génétique, Université de Paris, fall semester.)
Welshons, William J. A.B. Research assistant.
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Dempster, Everett R. Ph.D. Associate Professor. Radiation genetics and polygenic inheritance.

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Frye, Sara (Mrs.) B.A. Curator of Stocks.
Herskowitz, Irwin H. Ph.D. Research Executive. Mutation, developmental genetics.
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Muller, H. J. Ph.D. Professor.
Oster, Irwin I. B.S. Graduate student, Teaching Assistant. Developmental genetics.
Valencia, Juan I. Ph.D. Research Associate. Mutation, cytogenetics.
Valencia, R. M. (Mrs.) Ph.D. Research Fellow. Heterochromatic effects, mutation.
Verderosa, Fred. A.B. Graduate student, Research Assistant.
Wortman, Diana. A.B. Graduate student, Technical Assistant.

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Fogel, Seymour. Ph.D. Assistant Professor. Melanogaster: induced and spontaneous mutation studies.
Goodman, Fred. B.S. Graduate student. Melanogaster: crossing over.

Cambridge 38, Massachusetts
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Blumenheim, Ursel. Student. Persimilis: physiological properties of inversions.
Frisch, Rose (Mrs.) Ph.D. Melanogaster: developmental genetics.
Spiess, Eliot B. Ph.D. Instructor. Persimilis: population genetics.

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Costello, D. P. Ph.D. Professor and Chairman. Embryology, cytology, implantation, radiophosphorus.
Giles, Alice Ryland (Mrs. R. E.) M.A. Research Assistant. Melanogaster.
McMaster, Mary C. A.B. Graduate student. Melanogaster.
Whittinghill, M. Ph.D. Associate Professor. Irradiation, chemical mutagens, gonial and meiotic crossing over.

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Allen, Sally Lyman.
Buri, Peter F. Drosophila genetics.
Burkholder, John H. Drosophila genetics.
Foster, Morris.
Gersh, Eileen Sutton. Melanogaster.
Hill, Berton F.
Paulson, Richard E.
Silvers, Willys.
Spofford, Janice Brogue. Drosophila Genetics
Strandskov, H. H.
Wolff, George L.
Wright, Sewall.

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DeMarinis, F. Ph.D. Chairman. Gene action.
Hoch, Milton O. M.A. Lecturer. Chemical mutagens.

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Cold Spring Harbor, New York
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Buchanan, Jennie S. (Mrs. Paul) Research Assistant. Melanogaster.
Demerec, M. Ph.D. Director. Melanogaster.
Gay, Helen. M.A. Research Assistant. Melanogaster: electron microscopy of salivary chromosomes, chromosome organization, cytochemistry. (Present address: University of Pennsylvania.)
Kaufmann, B. P. Ph.D. Investigator. Melanogaster: cytology, cytochemistry.
Larrabee, Janice. B.A. Research Assistant. Melanogaster: cytogenetics, cytochemistry.
Smith, Guinevere C. (Mrs. W. W.) M.A. Curator of Stocks.

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Madden, Carol V. B.A. Research Assistant. Second-chromosome lethals.

McGunnigle, E. C. B.S. Research Assistant.
Wallace, Bruce. Ph.D. Geneticist. Population studies.

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Blue, J. Technician.
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Novitski, E. Ph.D. Associate Professor.
Sears, E. R. Ph.D. Principal Geneticist, USDA.
Singleton, J. R. Ph.D. Assistant Professor.
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Zimmering, S. M.A. Graduate assistant.

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Barish, Natalie. M.S. Graduate student. Immunogenetics.
Chovnick, Arthur. M.S. Research Assistant. Immunogenetics.
Feldmiller, Victor A. B.S. Graduate student. Biochemical genetics.
Fox, Allen S. Ph.D. Assistant Professor of Zoology. Immunogenetics, physiological genetics.
White, Thomas B. B.A. Research Assistant. Immunogenetics.
Wolf, Robert. B.S. Graduate student. Biochemical genetics.
Ziebur, Nancy Kent. Ph.D. Research Assistant. Induced mutations.

Davis, California
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Green, M. M. Ph.D. Assistant Professor.
Shapard, Pauline. A.B. Graduate student.

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Burns, Jean. A.B. Graduate student.
Clancy, C. W. Ph.D. Associate Professor.
Kubler, H. E. A.B. Graduate student.

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Northwestern University, Department of Biological Sciences

Blount, Jerry Lee. M.S. Research Assistant. Melanogaster: mutation, cytogenetics.
Mickey, George H. Ph.D. Associate Professor. Melanogaster: mutation, crossing over, cytogenetics.

Gambier, Ohio
*Kenyon College, Department of Biology

Power, Maxwell E. Ph.D. Associate Professor of Biology. Melanogaster: neurology.
Neidhardt, Fredrick C. Student Assistant. Melanogaster: behavior.

Hempstead, New York
Hofstra College, Department of Biology

Klein, Richard L. Graduate student. Melanogaster: lethal action.
Madden, Carol. Graduate student. Melanogaster: lethal action.

Marengo, Norman P. Ph.D. Assistant Professor. Prosaltans: development.
Meiselman, Newton. Graduate student. Prosaltans: development.
Prestiano, Robert. Graduate student. Prosaltans: development.
Reiter, Gloria R. Graduate student. Melanogaster: lethal action.
Warren, K. Brehme. Ph.D. Assistant Professor. Melanogaster: lethal action.

Houston, Texas
The Rice Institute, Department of Biology

Altenburg, Edgar. Ph.D. Associate Professor.
Altenburg, Luolin Sorey. Ph.D.

Houston 6, Texas
The University of Texas, M. D. Anderson Hospital, Department of Biology

Browning, Iben. M.D. Virilis.

Kingston, Rhode Island
University of Rhode Island, Department of Zoology

Hartung, E. W. Ph.D. Associate Professor. Melanogaster: factors influencing tumor incidence.
Jones, F. F. Technician.
Knutson, H. Ph.D. Professor. Melanogaster: effects of certain insecticides on reproductive rate and capacity.

Knoxville, Tennessee
The University of Tennessee, Department of Zoology and Entomology

Baker, William K. Ph.D. Assistant Professor. Radiation genetics, gene action, speciation. (On leave at Oak Ridge National Laboratory.)
Carpenter, John M. Ph.D. Assistant Professor. Drosophila populations, biotic potential and environmental resistance, Intra- and interspecific competition.
Hickey, John M., Jr. Graduate student. Gene action.

Lincoln, Nebraska
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Annan, Murvel E. M.S. Graduate student. Melanogaster: radiation.
Miller, Dwight D. Ph.D. Associate Professor. Affinis subgroup: mating behavior.
Weeks, Leo. M.A. Graduate student. Melanica.
Yanders, Armon F. M.S. Graduate student. Robusta: radiation.

Logan, Utah
Utah State Agricultural College, Department of Zoology

Gardner, Eldon J. Ph.D. Professor. Melanogaster: genetics.

Los Angeles, California
University of California

Ball, Francis M. B.S. Senior Laboratory Technician, curator of species stocks, Department of Botany. Pseudoobscura: mutants.
Epling, Carl. Ph.D. Professor, Department of Botany. Pseudoobscura: population genetics.
GoodSmith, W. M.A. Graduate student, curator of melanogaster stocks, Department of Zoology. Melanogaster: position effect and the brown locus.
Hinton, Taylor. Ph.D. Lecturer, Department of Zoology. Melanogaster: position effect, gene expression on chemically defined medium.

Mattoni, Rudolf H. T. B.S. Graduate student, Department of Zoology. Pseudoobscura: population genetics.
Mitchell, Donald F. B.A. Graduate student, Department of Zoology. Pseudoobscura: population genetics.
Queal, Marion. Ph.D. Research Associate, Department of Zoology. Melanogaster: mutants.
Roberts, Margaret. M.A. Laboratory Assistant, Department of Zoology. Melanogaster: gene expression on chemically defined medium.

Madison 6, Wisconsin
University of Wisconsin, Department of Genetics

Crow, James F. Ph.D. Associate Professor. Population genetics.
Seto, Frank. Graduate assistant. Embryology.
Smith, Daniel. Graduate assistant. DDT resistance.

Minneapolis 14, Minnesota
University of Minnesota, Department of Zoology

Kroman, Ronald K. Graduate student. Melanogaster.
Merrell, David J. Assistant Professor. Population genetics.
Reed, Sheldon C. Professor. Population genetics.

New Haven, Connecticut
Albertus Magnus College, Department of Biology

Cullen, Sister Mary Urban, O.P. Ph.D. Professor. Melanogaster: developmental genetics.

New Haven 11, Connecticut
Yale University, Osborn Zoological Laboratory

Bull, Alice L. M.A. Graduate student. Melanogaster: developmental genetics.
Gall, Joseph C. B.S. Graudate student. Cytology, chromosome structure, and physiology.
Hilse, Regina M. (Mrs.) B.A. Curator of stocks, technical assistant.
Poulson, D. F. Ph.D. Associate Professor. Melanogaster: developmental genetics; mineral metabolism of Drosophilidae.
Remington, Charles L. Ph.D. Assistant Professor. Speciation; population genetics of Lepidoptera.
Ross, Bertha G. B.A. Graduate student. Developmental genetics, histochemistry.

New London, Connecticut
Connecticut College, Department of Zoology

Wheeler, Bernice M. Ph.D. Instructor. Gibberosa: physiological genetics; iodine metabolism of Diptera.

New Orleans 12, Louisiana
Louisiana State University School of Medicine

Aitkens, Adrien. A.B. Research Assistant.
Burdette, Walter J. Ph.D., M.D. Director of Research and Teaching in Oncology, Associate Professor of Surgery. Tumors, mutations.
Forshag, Marjorie. B.S. Research Assistant.
Hyde, Harriette. B.S. Curator of stocks.
Nelson, Russell. A.B. Research Assistant.
Pope, Clare Lee. A.B. Research Assistant.

New York 24, New York

American Museum of Natural History, Central Park West at 79th Street

Mayr, Ernst. Ph.D. Population genetics.

New York 27, New York

Columbia University, Department of Zoology

Cooper, W. Postgraduate student. Population genetics and ecology, the role of different species of yeast in the diet of pseudoobscura and persimilis.

Cordeiro, A. R. Research Fellow of the Rockefeller Foundation. Willistoni: population genetics, lethals found in natural populations. Permanent address: Departamento de Genetics, Universidade de Porto Alegre, Porto Alegre, Brazil.

Dobzhansky, Th. Professor. Pseudoobscura, persimilis, willistoni, and other species: population genetics.

Dunn, L. C. Professor. Melanogaster: developmental genetics.

Kerr, W. E. Research Fellow of the Rockefeller Foundation. Population genetics, experimental studies on the genetic drift. Permanent address: Escola Superior de Agricultura, Piracicaba, Brazil.

Levine, Louis. Graduate student. Pseudoobscura: population genetics, heterosis.

Lewontin, Richard C. Postgraduate student. Pseudoobscura: population genetics, polymorphism.

Marien, D. Postgraduate student. Experimental studies on sexual isolation in Drosophila, population genetics.

Moore, John A. Professor. Population genetics, species competition.

Pavlovsky, O. A. Research Assistant.

Prout, Timothy. Graduate student. Melanogaster: population genetics, radiation genetics.

Rizki, M. T. M. Graduate student. Willistoni: developmental genetics, lethals.

Rosenbaum, Joan. Graduate student. Pseudoobscura; population genetics and ecology of Drosophila.

Spassky, Boris. Research Associate, Pseudoobscura, willistoni, and other species: population genetics, comparative genetics, mutation rates in different species.

Spassky, N. P. (Mrs.) Research Assistant. Willistoni, prosaltans: population genetics.

Townsend, J. T. Graduate student. Population genetics; marginal populations of willistoni and other species.

Vetukhov, M. A. Research Fellow. Population genetics and general genetics; heterosis and hybridization of local populations of pseudoobscura.

Norman, Oklahoma

University of Oklahoma, Department of Zoology

Blanc, Richard. Ph.D. Assistant Professor. Melanogaster: developmental genetics.

Oak Ridge, Tennessee

Oak Ridge National Laboratory, Biology Division

Alexander, Mary L. Ph.D. AEC Postdoctoral Fellow. Mutations in natural populations, and radiation genetics.

Baker, William K. Ph.D. Radiation genetics, position effect.

Sgourakis, Elizabeth. B.A. Radiation genetics.

Ward, Calvin L. Ph.D. AEC Postdoctoral Fellow. Speciation and radiation genetics.

Pasadena, 4, California
California Institute of Technology, Kerckhoff Laboratories of Biology

Beadle, G. W. Ph.D. Professor.
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Hinton, C. W. M.A. Graduate student.
Judd, B. H. M.A. Graduate student.
Lewis, E. B. Ph.D. Associate Professor.
Lindsley, D. L. M.A. Graduate student.
Morgan, L. V. (Mrs. T. H.) M.A. Research Associate.
Sturtevant, A. H. Ph.D. Professor.
Vogt, Marguerite. M.D. Gosney Fellow.

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Institute for Cancer Research and Lankenau Hospital Research Institute, Fox Chase

Bischoff, Norma. Technician.
Dackerman, Marjorie. A.B. Research Assistant.
Gunter, Walter. Laboratory helper.
Hungerford, David A. A.B. Technician.
Levenbook, Leonidas. Ph.D. Research Fellow.
Patterson, Elizabeth K. Ph.D. Associate Member.
Rudkin, George T. Ph.D. Research Associate.
Schultz, Helen Redfield. Ph.D. Research Fellow.
Schultz, Jack. Ph.D. Senior Member, Head of Department.

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Princeton University, Department of Biology

Anderson, Banks. Senior student. Ring chromosomes in Drosophila.
Cooper, K. W. Ph.D. Associate Professor. Drosophila cytogenetics.
Krivshenko, J. D.Sc. Research Associate. Busckii.

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King, James. B.S. Technical assistant.
Moree, Ray. Ph.D. Assistant Professor. General and population genetics; hybrid sterility.

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Hughes, Roscoe D. Ph.D. Professor of Biology. Drosophila cytogenetics.

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Bennett, Katherine W. A.B. Graduate student. Robusta: cytogenetics.
Blight, William C. A.B. Graduate student. Americana: cytology and ecology.
Carson, Hampton L. Ph.D. Associate Professor. Robusta: cytogenetics.
Romano, Albert. A.B. Graduate student. Americana: genetics and ecology.
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Hanks, George. B.A. Graduate assistant. Melanogaster: mutation.
Lefevre, George, Jr. Ph.D. Assistant Professor. Melanogaster: radiation genetics.
Newby, W. W. Ph.D. Professor and Department Head. Melanogaster: developmental genetics, tumorous head.
Ratty, Frank J., Jr. M.S. AEC Predoctoral Fellow. Melanogaster: lethals, position effect.
Stephens, F. E. Ph.D. Professor. Human genetics.

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Young, Frank. Student.

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Roman, Herschel. Ph.D. Associate Professor of Botany. Cytogenetics.
Zalokar, Marko. Ph.D. Assistant Professor of Zoology. Neurospora genetics.

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King, Robert C. B.S. Graduate student. Melanogaster: mutation studies with radioisotopes; phosphorus metabolism.
Ryan, Marion B. B.S. Curator of stocks.

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Bourgin, Rose Cherie (Mrs.) M.S. Radiation genetics.
Kurland, Aaron. Graduate student. Physiological genetics, Bar alleles.
Luce, Wilbur M. Associate Professor. Bar alleles, effects of environmental agents, radiation genetics, physiological genetics.
Shirley, Herschel V., Jr. Graduate student. Population genetics.
Swenson, Adela J. Student, curator of stocks, laboratory technician.

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Wellesley College, Department of Zoology and Physiology

Wilson, Louise Palmer. Ph.D. Associate Professor. Melanogaster: physiology of growth, emphasis on tumors.

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Rifenburgh, S. A. Ph.D. Associate Professor. Melanogaster: ultraviolet radiation.

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College of Wooster, Laboratory of Biology

Spencer, W. P. Ph.D. Professor. Drosophila species; population genetics.

Abrahamson, Seymour. U.S.A., Bloomington, Indiana
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Akita, Y. K. Japan, Tokyo
Alcobé, S. Spain, Barcelona
Alexander, Mary L. U.S.A., Oak Ridge, Tennessee
Allante, M. (Mrs.) France, Gif-sur-Yvette (Seine et Oise)
Allen, Sally Lyman. U.S.A., Chicago, Illinois
Altenburg, Edgar. U.S.A., Houston, Texas
Altenburg, Luolin Sorey. U.S.A., Houston, Texas
Altorfer, Nelly. Belgium, Brussels
Anders, Georges. Switzerland, Zuerich
Anderson, Banks. U.S.A., Princeton, New Jersey
Andrés, José Ma. Argentina, Buenos Aires
Anger, Renate. Germany, Hamburg-Eppendorf
Annan, Murvel. U.S.A., Lincoln, Nebraska
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Bagnol, Jeanine. France, Paris
Baker, Percy H. U.S.A., Baltimore, Maryland and Richmond, California
Baker, William K. U.S.A., Knoxville, Tennessee and Oak Ridge, Tennessee
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Becker, Hans-Joachim Germany, Göttingen
Belitz, Hans-Joachim. Germany, Berlin-Buch
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Bösiger, B. Switzerland, Basle
Bösiger, E. Switzerland, Basle
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Bourgin, Rose Cherie (Mrs.) U.S.A., Urbana, Illinois
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Brink, Vernon C. Canada, Vancouver, B. C.
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Burkholder, John H. U.S.A., Chicago, Illinois
Burla, Hans. Switzerland, Zuerich
Burns, Jean. U.S.A., Eugene, Oregon
Burtin, Genevieve. France, Paris
Buzzati-Traverso, A. Italy, Pavia and U.S.A., Berkeley, California
Byers, Helen Louise. U.S.A., Bloomington, Indiana

Carpenter, John. Great Britain, London, England
Carpenter, John M. U.S.A., Knoxville, Tennessee
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